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SILICOTIC AND TUBERCULOSILICOTIC LESIONS SIMULATING BRONCHIOGENIC CARCINOMA

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IT HAS long been known that patients suffering from pulmonary silicosis with or without an added tuberculous infection may develop areas of fibrosis which in chest roentgenograms appear as more or less massive areas of density. A number of writers have drawn attention to the fact that these densities may be mistaken for bronchiogenic carcinoma, developing on a background of silicosis. In 1947 Woodruff and Kelley[1] reported a case of tuberculosilicosis simulating bronchiogenic carcinoma and reviewed the literature. They were able to find eight other cases of histologically confirmed silicosis or tuberculosilicosis which had been diagnosed lung tumor erroneously on clinical and radiographic grounds. Their patient showed a unilateral circumscribed density in the right lower lobe, with radiographic evidence of a diffuse silicotic reaction; he had a long history of exposure to dust. The case of Bradshaw and Chodoff[2] had a history of exposure to silica but no evidence of a diffuse silicotic reaction. When their patient was seen first the shadow was unilateral, but over a period of seventeen months, while he was under observation, a density appeared in the opposite apex. In all the other cases reviewed by Woodruff and Kelley the densities were bilateral.

A number of patients have been seen at the Toronto General Hospital with gross evidence of silicosis who showed massive densities, either unilateral or bilateral, which could be confused with carcinoma, and some of them actually had carcinoma. However, special interest has been aroused by a group of eight patients who, with one exception, have not shown the usual radiographic picture of silicosis, and have presented with a unilateral density of the type usually seen in bronchiogenic carcinoma but due in fact to silicosis or tuberculosilicosis. These eight patients, who are the subjects of this report, were all men in middle or late middle age; seven of the eight complained of symptoms of the type commonly associated with bronchiogenic carcinoma. These seven have been

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operated upon, and the diagnosis has been confirmed by histologic study. All have had a history of some exposure to silica dust, but a striking feature is that in some of them the exposure had been relatively short and many years before the onset of symptoms.

The reason for the development of massive pulmonary densities in patients with silicosis has never been elucidated. At the various Saranac Laboratory Symposia on Silicosis there was much discussion of this problem. Gardner[3] gave it as his opinion that infection, and most often tuberculous infection, is the underlying cause of conglomeration, although associated action of nonsiliceous dusts probably plays an accessory role. He suggested that in simple conglomerate silicosis the infection had healed and played only an etiological role in causing the density, while in tuberculosilicosis the infection is still active and recognizable as a tuberculous process. He gave a very detailed discussion of the various types of reaction which might result depending on whether the infection preceded the dust exposure, was coincident with it, or followed it.

At the Fourth Symposium, Pendergrass[4] raised the question of whether or not a massive shadow seen in a roentgenogram may be considered due to silicosis in the absence of the classical picture of diffuse nodulation, and stated that in his opinion it could. Gardner[5] had stated earlier, "The massive type of shadow usually appears upon a background of generalized nodulation, but there are plenty of cases in which this background is lacking or obscured." He pointed out that in such cases examination at autopsy had usually revealed minute nodules in other parts of the lungs. Riddell[6] described a case in which the exposure to silica had been very heavy, but the radiographs had been diagnosed as indicative of tuberculosis. Autopsy had revealed no evidence of infection. There was a conglomerate focus of silicosis, and a few nodules were discovered in other parts of the lungs. Gardner[7] ended the discussion by saying, in spite of his statement quoted above, that before a massive shadow, seen in a roentgenogram, could be considered silicotic he must insist on seeing nodular shadows. He admitted that in following this rule mistakes would be made and injustice done to individuals.

This rather brief summary of the various discussions at the Saranac Laboratory Symposia, and in particular the Fourth, is given because, if our interpretation of the pathology is correct, the series of cases to be described below includes examples of massive densities due to both conglomerate silicosis and tuberculosilicosis in the absence of radiographic evidence of nodulation.

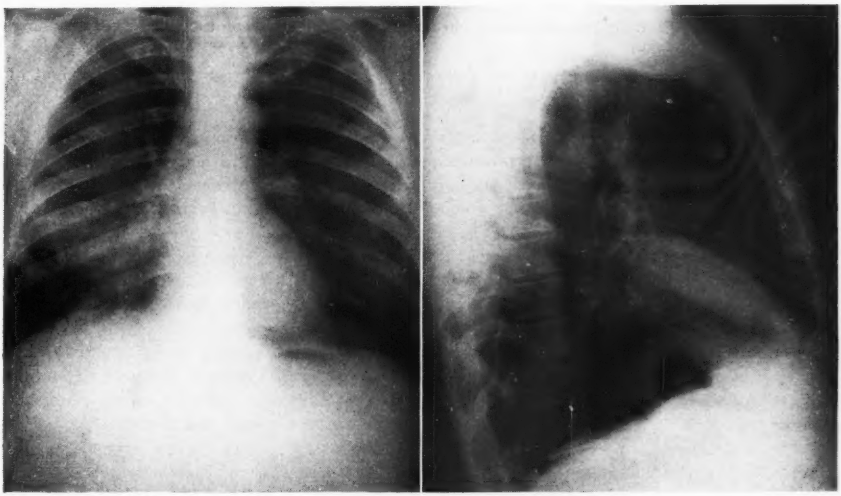
CASE REPORTS

CASE 1.—Mr. L. D., aged 49 years, was admitted to the Toronto General Hospital on Dec. 18, 1946, with a provisional diagnosis of bronchiogenic carcinoma. He had been well until two years previously when he began to lose energy and tire easily. For eighteen months he had had substernal pain radiating around the right costal margin, aggravated by a deep breath or cough. In June, July, and November, 1946, he had been treated in the hospital for attacks of right-sided pneumonia, and since the first attack in June he had had a cough productive of 15 c.c. per day of mucopurulent sputum. He had noticed blood in his sputum on two occasions during intervals between his attacks of pneumonia.

He had lost 30 pounds in weight, felt weak, and was short of breath on mild exertion. For twenty-six years this man had worked underground in hard rock gold mines as a pipe fitter and handling sand and cement to make underground walls.

He was a large man, weighing 185 pounds, who looked chronically ill. There were no abnormal signs in the chest or elsewhere on physical examination. Roentgenograms of the chest (Fig. 1) indicated partial atelectasis and consolidation of the right middle lobe. There was no evidence of silicosis. A bronchogram showed no filling of the middle-lobe bronchus, suggesting complete obstruction. Bronchoscopic examination revealed edema of the orifice of middle-lobe bronchus with mucoid secretion coming from the middle lobe. Repeated sputum examinations failed to show malignant cells.

It was concluded that the patient had obstruction of the middle-lobe bronchus, probably due to carcinoma, and secondary infection in the lobe.



A. *B.*

Fig. 1.—Case 1. *A* and *B*, Partial atelectasis and consolidation of right middle lobe.

At operation on Jan. 18, 1947, the lung was adherent to the chest wall. When freed, it showed a mottled gray-green appearance; scattered through it were firm nodules varying from 3 to 5 mm. in diameter. The middle lobe was about 50 per cent reduced in volume, solid, and dark gray in color. There was a thickening of all the hilar structures with very firm lymph nodes adherent to the vessels and bronchi. On elevating the consolidated middle lobe to examine the anterior aspect of the hilus, a very hard node which had been adherent to both the middle lobe bronchus and the anterior pulmonary vein came away with the lobe making a rent in the vein, with resulting severe hemorrhage. A pneumonectomy was done by dissection technique.

The patient developed an empyema due to streptococcus hemolyticus, which cleared with irrigations of the pleural space and instillation of penicillin. He was discharged from the hospital on March 28, 1947.

He has remained well since that time, but complains of shortness of breath on exertion. He receives compensation on the basis of total disability which appears to affect his desire to work. In April, 1950, he was readmitted for assessment. He had gained 35 pounds since his operation. His vital capacity was 1,660 c.c., or 57 per cent of normal. The arterial oxygen saturation at rest was 20.3 volumes per cent or 92.5 per cent of normal.

Pathologic Examination.—Gross: This confirmed the presence of multiple small discrete nodules throughout the lung and partial atelectasis with consolidation of the middle lobe. On section this lobe showed moderate dilatation of the bronchus, and the parenchyma was dark gray with islands of greenish brown tissue. All the structures of the hilus were densely adherent to each other, and the lymph nodes were enlarged, hard, and adherent. The dissection necessary after tearing the pulmonary vein had disturbed the relations about the middle-lobe bronchus, but it appeared probable that the bronchus had been compressed by an adherent node.

Microscopic: Microscopic examination confirmed that the discrete nodules were composed of whorls of hyaline material containing crystals which were demonstrated by the polarizing microscope. The middle lobe showed a very marked chronic inflammatory reaction, with dense fibrosis, fetalization of alveoli, and dense accumulations of chronic inflammatory cells. The hilar nodes showed a very marked chronic inflammatory reaction with typical silicotic nodules.

Diagnosis.—Chronic bronchiectasis, chronic interstitial pneumonitis, fibrosis of the lung, silicosis of the lung.

Incineration of the lung gave the following result: ash 2.32 per cent of dry weight of lung, silica 0.23 per cent of dry weight, silica 9.7 per cent of ash.

CASE 2.—Mr. A. M., aged 69 years, was referred to the Toronto General Hospital on April 2, 1948, with a diagnosis of bronchiogenic carcinoma. The patient had been perfectly well and able to do hard work until six months before admission, when for the first time he developed a cough. This had become frequent and severe, and increasingly more productive until he was bringing up 60 to 90 c.c. of thick dark sputum per day, never foul smelling or frankly purulent, and no blood was seen. Three weeks before admission he developed constant aching pain referred to the right scapula, not affected by breathing or posture. There had been a moderate decrease in exercise tolerance, but no weight loss. The patient had worked as a molder in a foundry for fifty-five years.

On examination the patient was small and wiry, weighing 122 pounds. The trachea was shifted slightly to the right; there were no enlarged lymph nodes in the neck or axillae. Chest expansion was 1¼ inches. The right chest posteriorly showed an area of diminished percussion note, decreased breath sounds, and some coarse and medium râles.

Roentgenograms of the chest (Fig. 2) in the posteroanterior view demonstrated a rather diffuse and irregular area of density, which in the lateral view was seen to be a clear-cut density, of a wedge shape, occupying the upper part of the superior segment of the right lower lobe, without evidence of atelectasis. Scattered through the left lung were three small calcified lesions. There was no nodulation of the type seen in silicosis. Bronchoscopic examination showed marked narrowing of the right main bronchus to one-third or one-fourth of the normal size, just below the origin of the middle-lobe bronchus. There was no evidence of tumor. Mucoid secretion was coming from the lower lobe. Multiple sputum examinations were negative for cancer cells.

It was thought that the most probable diagnosis was bronchiogenic carcinoma of the right lower lobe; thoracotomy was done on May 5, 1948. The right chest was opened by resection of the sixth rib. The lung was densely adherent to the chest wall. The superior segment of the lower lobe contained a hard, irregular mass 5 cm. in diameter which extended to the hilus, and on palpation seemed typical of carcinoma. About the hilus were many enlarged lymph nodes, some hard and gray, others large and black. One of the former was removed, and on frozen section showed only inflammatory reaction. Pneumonectomy was undertaken, but the nodes were so densely adherent to the hilar structures that the pulmonary artery was torn, necessitating opening the pericardium and ligature of the vessels within the pericardial sac.

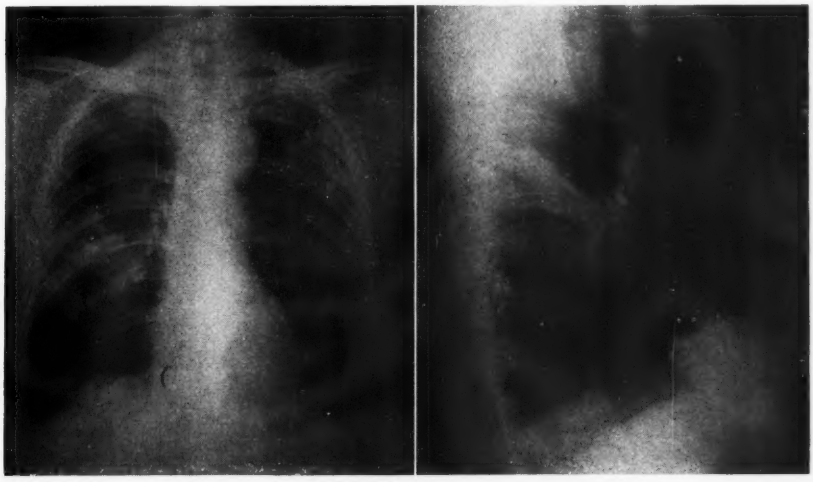
The patient made a good recovery from operation, but developed an empyema which healed satisfactorily with drainage and antibiotics. He was discharged from the hospital on June 4, 1948. On Feb. 4, 1951, he reported that his health was satisfactory, that he was unable to work, and was short of breath on slight exertion.

Pathologic Examination.—Gross: The superior segment of the lower lobe contained a firm mass which on section proved to be 5 by 4 cm. It was gray in color, with scattered anthracotic pigment, giving a mottled effect. The whole lung showed a considerable amount of anthracotic pigment.

Microscopic: Microscopic examination of the solid area demonstrated that it consisted of masses of fibrous tissue interspersed with fibrotic nodules showing a typical lamellar structure. The alveolar septa were thickened due to increased fibrous tissue and the presence of macrophages. The alveolae were mainly collapsed and contained pigment-bearing macrophages. Throughout the lung were many fibrotic nodules showing a concentric whorled appearance. The lymph nodes showed a similar fibrous tissue reaction.

Diagnosis.—Chronic interstitial pneumonitis (localized), and silicosis.

A portion of the lung was ashed with the following result: lung ash 8.28 per cent of dried weight, total silica 3.78 per cent of dried weight, total silica 45.6 per cent of the lung ash.



A. *B.*

Fig. 2.—Case 2. *A* and *B*, Atelectasis and pneumonitis involving upper part of superior segment of right lower lobe.

CASE 3.—Mr. W. H., aged 62 years, was admitted to the Toronto General Hospital on Feb. 1, 1949, with a provisional diagnosis of bronchiogenic carcinoma. Twelve months before admission he developed a dry cough, which two months later became productive of small amounts of yellow sputum, frequently streaked with blood. He occasionally had mild fever and sweating, lost energy, and had lost 21 pounds in weight. There was moderate shortness of breath on exertion. The patient was of Ukrainian birth, had lived in Canada twenty-two years, and worked as a construction laborer. At the age of 27 years he had worked for one year as a sand blaster.

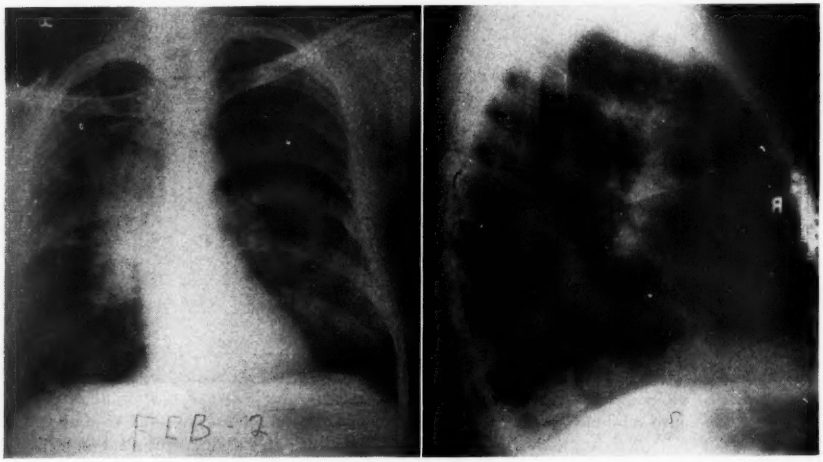
The patient appeared chronically ill. He had a frequent harsh cough productive of yellow-green sputum which was not foul. The trachea was midline, and there were no enlarged nodes in the neck. The right upper chest anteriorly showed decreased movement, dullness to percussion, and reduced breath sounds. Roentgenograms (Fig. 3) showed a large, irregular area of density extending out from the right hilus into the upper and

middle lobes; the shadow of the left hilus was increased, but there was no evidence of silicotic nodulation. Bronchoscopic examination showed a general mild inflammatory reaction of the right bronchial tree with no evidence of tumor. Laminograms showed a very dense mass surrounding and narrowing the right upper lobe bronchus, with the characteristic appearance of a bronchiogenic carcinoma. Multiple sputum examinations were negative for malignant cells.

The provisional diagnosis was bronchiogenic carcinoma of dubious operability.

Right thoractomy was performed on Feb. 15, 1949. The upper and middle lobes were densely adherent to the parietal pleura. A hard irregular mass appeared to arise at the upper lobe bronchus and fill the central portion of the upper lobe and to extend across an incomplete fissure into the middle lobe. Throughout the remaining portions of the lung were scattered many small discrete densities, up to 0.5 cm. in diameter. In the hilus were many very firm, gray, adherent nodes up to 1.5 cm. in diameter. One was removed, examined by frozen section, and reported to show "old tuberculosis and anthracosis, no evidence of tumor." A dissection pneumonectomy was done; this was difficult because of the nodes which were densely adherent to the great vessels and bronchus.

The patient made a good recovery and was discharged eighteen days after operation. He has been seen regularly since, the last time in September, 1951, and has remained well, with no cough, sputum, or hemoptysis, has gained 30 pounds, and has fair exercise tolerance.



A. *B.*

Fig. 3.—Case 3. *A* and *B*, Massive density involving right upper and middle lobes.

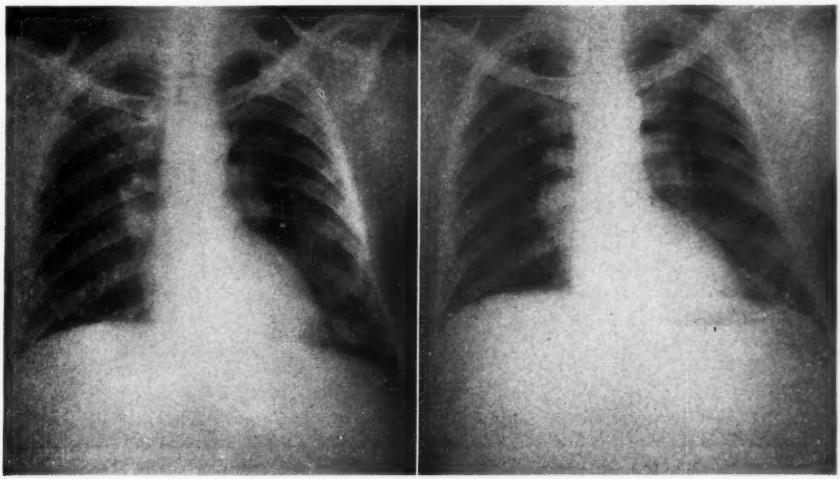
Pathologic Examination.—Gross: Section of the lung confirmed the presence of a large irregular mass 8 cm. in diameter in the upper lobe, extending into the middle lobe. The cut surface was light gray mottled with black. This mass appeared to compress the origin of the upper-lobe bronchus, but the more distal portions of this bronchus were dilated and contained mucous casts. The lymph nodes were very hard and enlarged up to 2.5 cm. in diameter; some were light gray on section, flecked with black, others were softer and jet black.

Microscopic: Microscopic examination showed that the dense areas consisted of a heavy deposit of fibrous tissue, with an infiltration of chronic inflammatory cells, mixed with nodules composed of concentric layers of hyaline material. Many doubly refractile crystals were present. The upper-lobe bronchus showed typical bronchiectasis. The lymph nodes showed dense fibrosis with silicotic nodules.

Diagnosis.—Silicosis of lung and lymph nodes, chronic interstitial pneumonitis, and bronchiectasis of the upper lobe.

CASE 4.—Mr. J. C., aged 57 years, was admitted to the Toronto General Hospital on July 9, 1951. He had no complaints and was referred because of a shadow in the right lung discovered on routine radiography. He had had regular examinations of his chest since 1945, and in 1949 a shadow was noticed radiating out from the right hilus (Fig. 4, *A*). Investigation at another hospital in January, 1950, included bronchoscopy. No evidence of carcinoma was found, and no definite diagnosis was made. Further films (Fig. 4. *B*) appeared to indicate an extension of the shadow, and he was referred for reinvestigation. The patient had no cough, no history of hemoptysis, no chest pain, no weight loss, and good exercise tolerance. He had worked in a stove and furnace foundry for thirty-five years, and had had some exposure to dust.

He looked perfectly well. The only abnormal finding was slightly decreased percussion note at the right base posteriorly.



A. *B.*

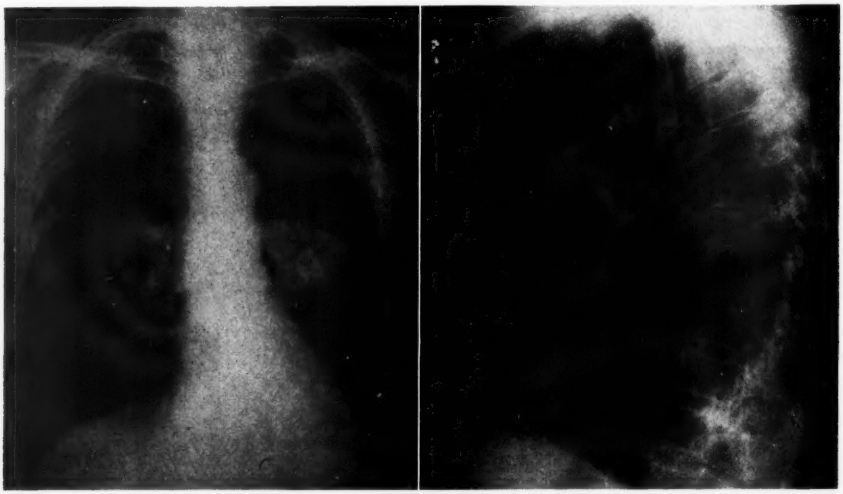
Fig. 4.—Case 4. *A*, Roentgenogram in 1949 showing density at right hilus with indefinite outline. Left hilar shadow increased. *B*, Roentgenogram eighteen months later shows right hilar shadow more dense. Lateral view demonstrates that it is in superior segment of lower lobe.

The roentgenograms showed increased hilar shadows with some nodulation which could be interpreted as evidence of minimal silicosis. The 1949 film showed a rather dense mass about 4 cm. in diameter with a fuzzy outline, lying at the level of the right hilus. Further films on this admission including laminograms and a bronchogram demonstrated that it lay in the superior segment of the lower lobe, that it had not increased appreciably in size, but the outline had become more clear-cut, and that the two divisions of the bronchus to the superior segment were somewhat distorted but not obstructed. Bronchoscopic examination and examination of secretions aspirated from that bronchus were all negative for malignant cells. Sputum cultures were negative for tubercle bacilli.

A diagnosis of conglomerate silicotic lesion was made. It was decided that in view of the lack of change in the lesion over a period of two years, and the negative results of bronchoscopic and cytological examinations, bronchiogenic carcinoma could be ruled out. Because of the lack of symptoms or disability of any kind, and beause we have learned that local resection of lung tissue in these cases is difficult, and frequently impos-

sible, it was decided not to undertake thoracotomy. His physician was advised to continue to keep the lesion under observation by repeated radiographs and to have further sputum cultures done.

CASE 5.—Mr. Wm. V. A., aged 58 years, was referred to the Toronto General Hospital on Dec. 19, 1951, with a provisional diagnosis of bronchiogenic carcinoma. He had been in good health until October, 1951, when he developed pneumonia. With treatment by antibiotics he made a slow recovery, but continued to have a cough with a small amount of mucopurulent sputum, lost 12 pounds in weight, and tired easily. There was moderate shortness of breath on exertion. During the pneumonia his sputum was streaked with blood. He was referred because of a persisting density in the left lower lobe on radiographic examination. For many years he worked in smelters and foundries, and had had much exposure to dust.



A *B*

Fig. 5.—Case 5. *A* and *B*, Diffuse density in superior segment of left lower lobe.

The patient looked older than the stated age, but was in good general condition. There was a small area of dullness with diminished breath sounds below the angle of the left scapula. Roentgenograms (Fig. 5) showed a diffuse area of density in the left midlung field, which in the lateral film was shown to occupy the upper part of the superior segment of the lower lobe. A lipiodol bronchogram showed failure of filling of the left superior segmental bronchus. A bronchoscopic examination showed some narrowing and distortion of the left lower-lobe bronchus without evidence of tumor. Aspirated secretions and three specimens of sputum were negative for malignant cells. Vital capacity was 3,500 c.c., or 80 per cent of the calculated normal.

In view of the long history of exposure to dust, and the lack of evidence of carcinoma on bronchoscopic and sputum examination, it was considered that the most probable diagnosis was obstructive pneumonitis secondary to bronchial obstruction by silicotic nodes. It was decided to do a thoracotomy to rule out carcinoma, and in the hope that the condition could be treated by lobectomy.

On Dec. 26, 1951, a left thoracotomy was done. The whole left lung showed a heavy deposition of anthracotic pigment, and a few scattered hard nodules 3 or 4 mm. in diameter could be palpated. The upper lobe was voluminous, and the lower slightly reduced

in volume. The superior segment was filled by an irregular firm mass, and was densely adherent to the parietal pleura. There was moderate thickening about the hilus, with many hard black adherent nodes, up to 1.5 cm. in diameter. A dissection lobectomy of the lower lobe was accomplished with difficulty, and in doing so it was necessary to free a collar of these nodes which surrounded and compressed the superior segmental bronchus at its origin. The lobe was immediately examined by frozen section, and no evidence of carcinoma was found.

The patient recovered well, and was discharged from the hospital after eleven days. At that time he was free from cough.

Pathologic Examination.—Gross: Section of the lung showed that in addition to the collar of hard nodes surrounding the superior segmental bronchus, there was a similar hard black node adherent to the bifurcation into the superior and inferior divisions of this bronchus, distorting and narrowing both of the divisions. In the superior segment was an irregular firm mass, gray in color, flecked with black areas, and becoming necrotic in the center.

Microscopic: Microscopic examination revealed that there was widespread necrosis in the firm area noted in the gross. The immediately adjacent lung showed a marked fibrous tissue thickening of the alveolar walls, infiltration by acute and chronic inflammatory cells, and many macrophages lying within the alveoli. The lymph nodes showed a heavy deposit of anthracotic pigment, fibrosis, and silicotic nodules.

Diagnosis.—Pulmonary abscess, diffuse interstitial fibrosis, and anthracosilicosis of lymph nodes.

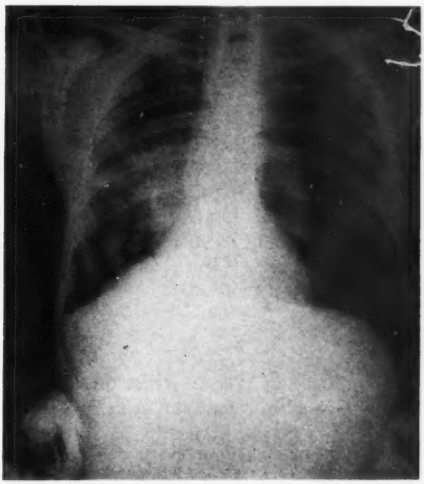


Fig. 6.—Case 6. Diffuse nodulation typical of silicosis, triangular shadow at right base indicating atelectasis of right lower lobe, and diffuse density in right mid-lung field.

CASE 6.—Mr. L. S., aged 49 years, was referred to Dr. N. S. Shenstone on Nov. 23, 1945. He had been a grinder and chipper in a foundry for more than twenty years. In 1944 a routine roentgenogram showed early diffuse nodulation, indicating silicosis, and he was advised to change his occupation, which he did not do. For several years he had been losing weight and strength, but was otherwise well until January, 1945, when he developed an acute febrile illness which was diagnosed as lobar pneumonia affecting the right lower lobe. He gradually recovered but continued to have cough and sputum, occasionally blood streaked, and the abnormal signs persisted at the right base. This led to a further roentgenogram (Fig. 6). This film showed diffuse nodulation typical of early silicosis, but in addition there was a shift

of the mediastinum to the right, a triangular density at the right base indicating atelectasis of the lower lobe, and an irregular area of density extending upward from the level of the hilus into the upper lobe area.

When referred nine months later there was no significant change in the radiographic picture. A right bronchogram confirmed the presence of an obstruction near the origin of the lower-lobe bronchus, with atelectasis of the lower lobe, and a deformity of the middle-lobe bronchus apparently due to a mass of lymph nodes at the hilus. A bronchoscopic examination showed a stenosis of the bronchus to the lower lobe without evidence of tumor. The bronchus contained bloody secretion. Sputum examinations were negative for the tubercle bacillus on smear.

It was concluded that the patient had pulmonary silicosis, complicated by bronchiogenic carcinoma, or possibly by tuberculosis, and thoracotomy was advised.

At operation on Feb. 12, 1946, by Dr. Shenstone, it was found that there were only a few pleural adhesions. The right lower lobe was atelectatic and extremely hard; continuous with it was a hard mass occupying the hilus of the lung and extending into the posterior part of the upper lobe. The overdistended middle lobe and anterior portion of the upper lobe contained many hard nodules like small peas. A dissection pneumonectomy was done.

The patient made a good recovery from the operation, and was discharged from the hospital eleven days later. It is reported by his doctor that he remains well, but is short of breath on exertion. Roentgenograms at frequent intervals have shown no significant change in the silicotic reaction in the left lung.

Pathologic Examination.—Gross: Extending from the hilus into the posterior segment of the upper lobe and filling the atelectatic lower lobe was a hard mass, blue-black on section. This mass surrounded the bronchi, reducing them to slits. In the lower lobe was an abscess 1.5 cm. in diameter.

Microscopic: Sections from the small discrete nodules and from the main mass showed a typical silicotic reaction, with dense areas of hyaline material. The center of some of the nodules showed necrosis. The abscess cavity showed caseation with destruction of part of a bronchial wall. In some areas the bronchi were compressed by a dense silicotic fibrosis, and in other areas they were dilated and showed tuberculous ulceration.

Diagnosis.—Silicosis and secondary tuberculosis.

CASE 7.—Mr. S. S., aged 63 years, was admitted to the Toronto General Hospital on Oct. 25, 1948. This retired postman had been well until July, 1948, when he began to notice increasing fatigue and weakness. In September he began to cough and raise a few cubic centimeters of yellow sputum, but no sign of blood. About the same time he noticed a huskiness of the voice and a desire to clear his throat repeatedly. He developed a mild ache in the right anterior chest, and noticed increasing shortness of breath on exertion. He had lost 15 pounds since July. The patient had been a postman for thirty-eight years. It was only after evidence of silicosis had been found at operation that direct questioning revealed the fact that he had been a driller in the silver mines at Cobalt for two years before entering the postal service.

He did not appear ill. The trachea was deviated to the right, and there was dullness with diminished air entry over the right chest anteriorly. The vocal cords moved normally. Roentgenograms (Fig. 7) demonstrated a wedge-shaped density occupying the anterior segment of the right upper lobe. Laminograms showed large rounded densities in the right hilus and under the carina suggestive of enlarged nodes. There was no evidence of silicosis. Bronchoscopic examination showed widening and fixation of the carina. Purulent material issued from the right upper-lobe bronchus, the middle-lobe bronchus was patent, but just below this there was a uniform stenosis of the lower-lobe bronchus to 4 mm. Sputum and aspirated secretions were negative for malignant cells and for tubercle bacilli on smear.

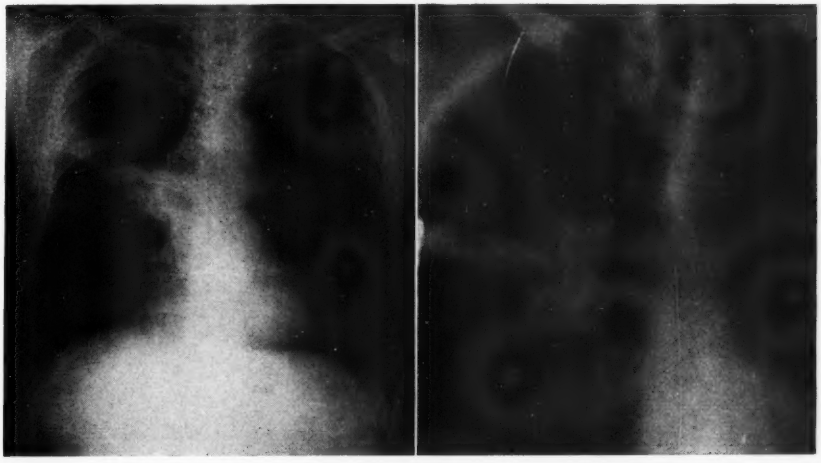
Bronchiogenic carcinoma was considered the most likely diagnosis, although the demonstration of many markedly enlarged nodes about the hilus suggested the possibility of obstructive pneumonitis due to bronchial compression by a node.

At operation on Nov. 2, 1948, the right chest was opened. The upper lobe was adherent by vascular adhesions. The anterior segment of the upper lobe was occupied by a very hard

mass which ran into the hilus. The whole bronchovascular tree running down into the lower lobe was markedly thickened, stiff, and irregular. Many enlarged nodes surrounded the hilus and were densely adherent; some were smaller, gray, and hard, others were large, soft, and jet black. Under the carina was a node as large as a big chestnut, black, and soft. These findings were considered typical of a silicotic reaction in the nodes, but one could not be certain that there was not a carcinoma also. Lobectomy was impossible because of the exceedingly dense reaction about the vessels. A dissection pneumonectomy was done with some difficulty.

The patient made a good recovery and was discharged ten days after operation. He has remained well, has regained his normal weight, has a fair exercise tolerance, and does light work.

Pathologic Examination.—Gross: Cutting the lung showed that the very hard consolidation of the anterior segment of the upper lobe was a dark, mottled, slate gray in color. There were similar smaller areas scattered through the upper and lower lobes, which were both deeply pigmented, but the middle lobe was crepitant and pink in color. Dissection of the upper-lobe bronchus showed that a hard, black node 1.5 cm. in diameter was densely adherent to the anterior segmental bronchus, compressing it and eroding the wall. Other similar nodes were adherent to the lower-lobe and middle-lobe bronchi.



A. *B.*

Fig. 7.—Case 7. *A* and *B*, Marked deviation of trachea to right and density indicating atelectasis of anterior segment or right upper lobe. Laminogram shows the marked reaction around the upper lobe bronchus and the shadow of large lymph nodes at the hilus.

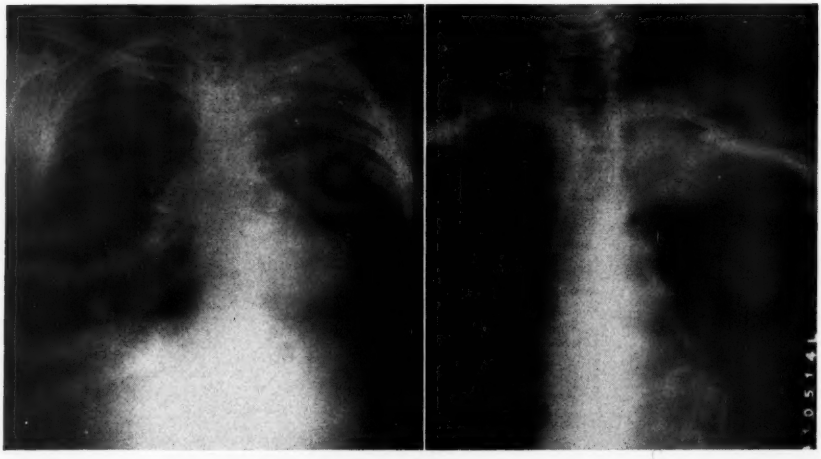
Microscopic examination of the area of consolidation showed that it consisted of dense areas of fibrosis with many scattered hyaline nodules. There was an infiltration with chronic inflammatory cells, and the alveoli were compressed and filled with exudate and macrophages. There were scattered areas of caseation. The lymph nodes showed a dense laminated fibrous tissue reaction with heavy deposits of carbon pigment. There was no evidence of tuberculosis in these nodes.

Diagnosis.—Tuberculosilicosis.

CASE 8.—Mr. B. C., aged 54 years, was admitted to the Toronto General Hospital on May 24, 1951. His first chest symptom was a febrile illness in the autumn of 1949 which was described as influenza. He had fever and was in bed for seven days. Roentgenograms taken at that time showed a diffuse infiltration in the left upper lobe. A film one month later showed

that it had cleared. He remained perfectly well, with no cough or sputum, until December, 1950, when he had a similar febrile attack and spent ten days in bed. He again recovered completely and became free of cough. In April, 1951, a routine chest film showed an area of density in the left upper lobe and he stopped work. During the month before admission, while he was not working, he gained 12 pounds. For several years he had noticed moderate shortness of breath on exertion. This man had been a miner since the age of 16 years. From 1920 until 1930 he worked underground in a hard rock gold mine where the incidence of silicosis is high.

Examination showed a large, powerful man weighing 230 pounds. The only significant finding was slight reduction in air entry into the left upper lobe posteriorly. Roentgenograms (Fig. 8) demonstrated a wedge-shaped area of density occupying the apicoposterior segment of the left upper lobe. A laminogram showed a very dense area in the extreme apex, and below this a large triangular-shaped density with a very clearly defined margin. The hilar shadows on both sides were greatly increased in bulk and density, but there was no diffuse nodulation. A bronchoscopic examination showed edema and reddening about the orifice of the left upper-lobe bronchus. No biopsy was obtained but secretions were aspirated. This specimen and three specimens of sputum were negative for malignant cells. No acid-fast bacilli were seen on smear.



A. *B.*

Fig. 8.—Case 8. *A* and *B*, Diffuse density at the left apex and marked increase in bulk and density of right hilar shadow. Laminogram shows a very dense shadow at the apex with a less dense triangular shadow below.

In view of this patient's occupational history and the other evidence, it was thought that the density represented a conglomerate silicotic lesion, but carcinoma could not be excluded. It was decided to do a thoracotomy and, if the diagnosis was confirmed and carcinoma could be ruled out, to perform a lobectomy. If this was not possible for technical reasons, it would not be desirable to do a pneumonectomy, because the disability did not justify the severe impairment of respiratory function which would result in this very big man whose vital capacity was 2,902 c.c. or 55 per cent of the calculated normal.

At operation on June 1, 1951, the left chest was opened. The apicoposterior segment of the upper lobe was filled by an extremely hard mass and was very densely adherent to the parietal pleura. The lobe was freed. Scattered through the lower lobe and anterior part of the upper lobe were hard nodules 2 to 3 mm. in diameter; one was excised. On section it was jet black; frozen section showed a typical silicotic lesion. About the hilus of the lung

there was a dense fibrosis and many gray stony hard nodes, up to 1.5 cm. in diameter, were adherent. The bronchovascular tree to both upper and lower lobes was greatly thickened, irregular, and stiff. It was decided that a dissection lobectomy was impossible, but a pneumonectomy could be done. The main density was cut into and a generous biopsy was removed. The cut surface was black. Frozen section showed silicosis. The chest was closed.

Further study of the sections confirmed the diagnosis of silicosis with no evidence of tuberculosis. The polarizing microscope revealed many doubly refractile crystals.

The patient recovered well and was discharged from the hospital twelve days later. One month after his operation the patient felt well and was anxious to return to work as a miner, but a report was received that a sputum culture planted two months previously had grown a typical tubercle bacillus. In August, 1951, he had a recurrence of pneumonia and further sputum cultures were done, of which three were positive for tubercle bacillus. He was admitted to the Toronto Hospital for Tuberculosis on Sept. 6, 1951.

This patient undoubtedly has tuberculosilicosis, and illustrates the difficulty of recognizing the presence of tuberculosis in a massive density even when biopsy is done. It is probable that he will require resection to control his tuberculosis.

SUMMARY OF CASES

The table summarizes the symptoms of which these patients complained. The oldest patient was 69 years of age and the two youngest were 49, with an average age of 58 years. Five had had prolonged exposure to dust, three as foundry workers, one as a grinder, and one as a miner. Of the other three, one had worked underground in a mine without direct exposure, one had been a sandblaster for a year, thirty-five years previously, and one had been a driller in a hard rock mine for two years, thirty-eight years previously.

TABLE I

CASE NO.	AGE	PNEUMONITIS	SPUTUM	HEMOPTYSIS	CHEST PAIN	LOSS OF WEIGHT (LB.)	SHORTNESS OF BREATH
1	49	+ +	+	+	+	30	+ +
2	69	0	+ +	0	+	0	+
3	62	+ +	+ +	+ +	+	21	+
4	57	0	0	0	0	0	0
5	58	+	+	+	0	12	+
6	49	+	+	+	0	15	+ +
7	63	0	+	0	+	15	+ +
8	54	+ +	0	0	0	0	+

The right lung was involved in six patients and the left in two. The lobes and segments involved were as follows: right middle lobe, superior segment of right lower lobe, right upper and middle lobes, superior segment of right lower lobe, superior segment of left lower lobe, right upper and lower lobes, anterior segment of right upper lobe, and apicoposterior segment of left upper lobe.

Seven of the patients were subjected to thoracotomy and in five a dissection pneumonectomy was done, in one a dissection lobectomy, and in one biopsies were taken. A thorough examination of the excised lung has failed to show evidence of tuberculosis in four patients. In two others the lung showed definite tuberculosilicosis. The patient who had biopsies only failed to show tuberculosis in the sections, but his sputum was positive on culture; he was considered to have tuberculosilicosis. In the patient who has not been operated upon, the

diagnosis has been made on clinical grounds, and, since sputum cultures are negative for the tubercle bacillus and the lesion appears stationary, he is considered to have silicosis.

DISCUSSION

The picture found at thoracotomy in this group of patients is characteristic, sufficiently so that in the later cases it was easily recognized even when a history of exposure to dust had not been obtained and there was little reason to question the preoperative diagnosis of bronchiogenic carcinoma. The gross appearance of conglomerate silicosis and tuberculosilicosis may be identical; the distinction can be made only by careful microscopic study. Typically, the massive density has a segmental distribution, and this is fairly accurately maintained. The involved segment may retain its normal volume or be somewhat reduced, but in only one case was there complete atelectasis. The area is usually adherent to the parietal pleura by adhesions which are thick and avascular. The main mass is slightly irregular and stony hard, not rubbery. If it is cut into, the surface is black to dark gray, with a mottled or marbled appearance. The rest of the lung is usually rather voluminous, shows more anthracotic pigment than usual, and on palpation one can feel a few or many hard nodules, usually three or four millimeters in diameter. The lung root shows a characteristic type of chronic inflammatory reaction with many enlarged lymph nodes firmly bound to the vessels and bronchi by very dense fibrous tissue. Palpation reveals that the whole bronchovascular tree, including that to the uninvolved lobe, is thickened, irregular, and stiff; it resembles the gnarled and twisted limb of an old tree. The enlarged lymph nodes vary from one to two centimeters in diameter and are of two types, both commonly seen in the same patient. Some are very hard, and light gray externally; when cut they show white or light gray areas mottled with anthracotic pigment. Other nodes are smaller, softer, rupture easily, and are jet black.

Gross examination of the lung after removal has confirmed the segmental distribution of the massive density. Dissection of the bronchus to the involved segment has shown in most cases that a node is compressing and narrowing it but not obstructing it completely. In one case a hard node was actually eroding the bronchial wall. The bronchi in the involved segment are deformed, showing dilatation in some areas, and in others they are compressed by the surrounding dense tissue; the mucosa is inflamed, and in some cases has shown ulceration.

Microscopic examination demonstrates that the massive densities from different cases, or even different areas in the same specimen, show varying degrees of a chronic inflammatory reaction. In some there is thickening of alveolar walls with marked reduction in the size of the alveoli, and in others this has progressed to complete disappearance of alveoli and replacement by masses of fibrous tissue interspersed with groups of pigment-containing macrophages. Scattered through all areas is a sprinkling of inflammatory cells, which tends to be more acute in the earlier stages, with focal areas of suppuration, and of a more chonic type in the later stages of the reaction. The fibrosis is particularly

marked about the bronchovascular bundles; the bronchi are compressed, the vessels show a definite sclerosis, and the lymphatic channels are obliterated or choked by macrophages filled with black pigment. Mixed with the fibrotic areas are masses of laminated hyaline material, characteristic of a silicotic reaction. The small firm nodules scattered through the lung are typical silicotic

Fig. 9.

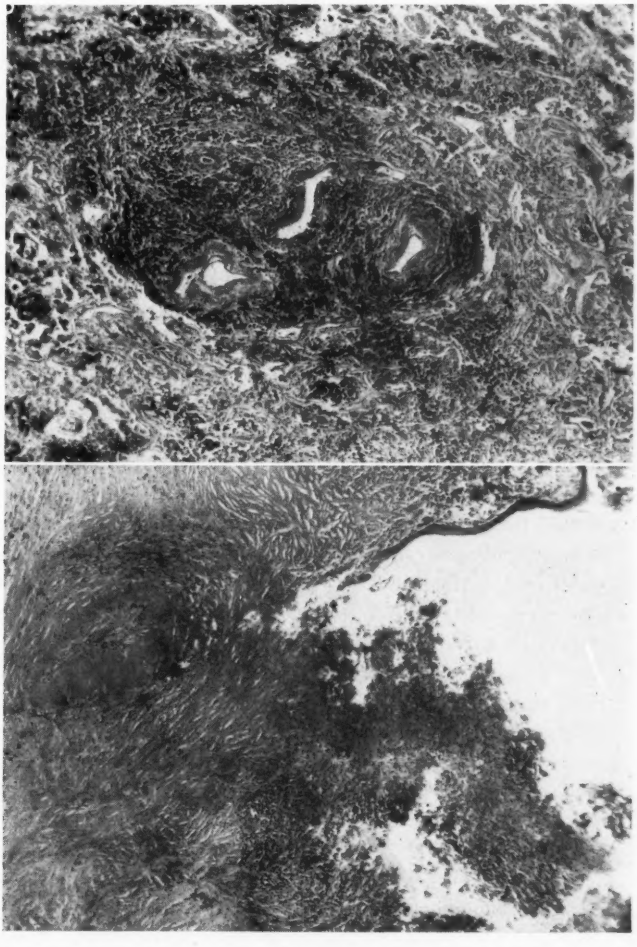


Fig. 10.

Fig. 9.—Case 2. A bronchovascular area showing the dense fibrous tissue reaction, a small bronchus which appears to be compressed, and sclerosis of the vessels. The lymphatic channels are filled with pigment-laden macrophages. The alveolar walls are thickened by fibrous tissue which is infiltrated with inflammatory cells of chronic type. (×35.)

Fig. 10.—Case 6. Tuberculosilicosis. A large bronchus shows metaplasia of the epithelium, with ulceration and caseation necrosis. At the left is a hyaline mass typical of a silicotic reaction. (×24.)

nodules. The hard gray lymph nodes are made up largely of masses of hyaline material, and mature fibrous tissue; the black nodes show silicotic nodules, fibrous tissue, and masses of macrophages filled with pigment.

The cases of tuberculosilicosis have shown a background picture like that just described, but added to it are more or less extensive areas of caseation, usually grouped about the bronchi. A point of interest is that the nodes which were adherent to and compressing the segmental bronchus showed a silicotic but not a tuberculous reaction.

From a study of the history of these patients, their clinical signs and the succession of changes shown in the serial roentgenograms which were available in some cases, together with the gross and microscopic examination of the excised tissue, it is possible to formulate a theory of the pathogenesis of the massive densities. There has been a growing realization among surgeons and pathologists that chronic bronchial obstruction, be it partial or complete, may lead to a chronic inflammatory process in the affected segment of lung, and that the picture seen varies from case to case depending on the duration and degree of the obstruction, the rate at which it has progressed, and the number of phases of acute or subacute infection which have occurred in the lung parenchyma. If the obstruction has developed quickly, before there has been much infection, atelectasis is a marked feature; if the obstruction progresses slowly and recurring infection has fixed the lung by fibrosis, there is little atelectasis, but bronchial damage is severe, resulting in secondary bronchiectasis. Paulson and Shaw[8] described this condition as it is seen in the middle lobe, and remarked that chronic atelectasis and pneumonitis may occur in any portion of either lung. They stated that a chronic pneumonitis develops as a result of atelectasis. The author is more inclined to believe that they are both due to the same cause, namely bronchial obstruction, and that the degree of either is variable. McDonald, Harrington, and Clagett[9] took this view and preferred to use the term obstructive pneumonitis for the condition; this term does seem to sum up better than any other the essential factors in this particular form of lung infection. They recognized clearly that to produce the characteristic changes in lung the bronchial occlusion need not be complete. In our own material we have found examples of chronic pneumonitis of segmental distribution due to bronchial compression by tuberculous nodes, sarcoid, nonspecific lymphadenitis (middle-lobe syndrome), and carcinoma. The involved lung in each case has shown a similar type of dense fibrosis with secondary bronchiectasis.

It is suggested that the so-called conglomerate lesions of silicosis are areas of obstructive pneumonitis due to partial or complete bronchial occlusion from involvement of segmental bronchi in a dense fibrous tissue reaction laid down around the lymph nodes, which are themselves the site of a silicotic reaction. Some of the effect may be due to actual mechanical compression of the bronchus, some due to physiologic obstruction by fixation of the bronchial wall, and some of the changes in the lobe or segment may be due to lymphatic obstruction. All the lymph drainage from a lobe of lung must filter through

the lymphatic channels and nodes in the lobar hilus, and in the presence of silicosis the resulting dense fibrosis of both the nodes and the peribronchial region must cause some degree of lymphatic obstruction. The partial bronchial obstruction determines the localization of infection in the affected segment, the chronic lymphatic obstruction with lymphedema prevents resolution of the inflammatory reaction and leads to the fibrosis typical of "unre-

Fig. 11.

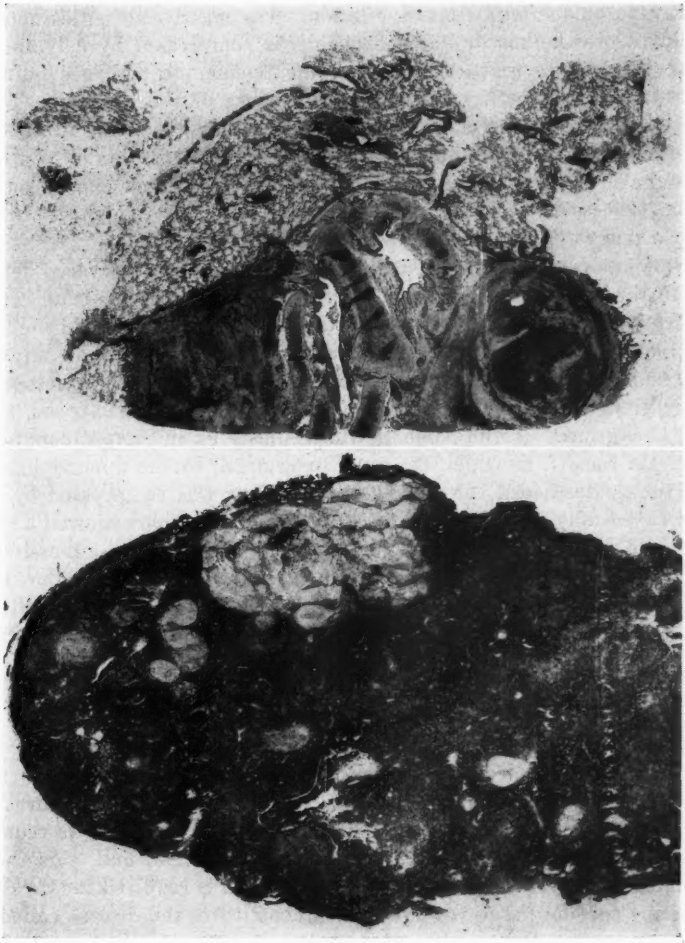


Fig. 12.

Fig. 11.—Case 7. A bronchus at its bifurcation compressed between two lymph nodes which show a silicotic reaction, but no evidence of tuberculosis. (×8.)

Fig. 12.—Case 5. A hard black lymph node, packed with pigment-laden macrophages, and showing nodules composed of whorls of hyaline material which are typical of a silicotic reaction. (×8.)

solved pneumonia." In silicosis, unlike carcinoma, the bronchial obstruction is relatively nonprogressive, so that the end result is a very chronic process in lung—what might be called end-stage obstructive pneumonitis.

If this explaination is correct, the importance of the silica as a cause of massive densities is mainly its effect on the lymph nodes. This explains some of the anomalies of the occurrence of massive densities, why many patients who have very obvious diffuse nodular shadows never show massive densities, while others who have no recognizable stigmata of silicosis in the roentgenogram may go on to form a massive lesion. The massive densities depend on the accident of whether or not a bronchus is compressed by a lymph node. The silicotic nodules scattered through the pneumonic area are quite inert and serve chiefly to betray the underlying cause of the reaction. The fact that they are more numerous in the dense area than elsewhere in the lung is due to the contraction of the segment pulling them together into a smaller space, and also in those patients who have continued to have exposure to silica after the process has started, to interference with the lymphatic drainage, through which foreign bodies are removed normally.

A very interesting question, and one which cannot be answered definitely, relates to the place that tuberculosis plays in this picture. Is it an early factor, which perhaps contributes to the lymph node enlargment and the fibrosis, or is it a late invader which finds a favorable medium in an area already fibrotic as the result of recurrent nonspecific infection? A study of these specimens certainly gives the impression that the tuberculosis is superimposed, that the fibrotic background is the same in tuberculosis as in simple conglomerate silicosis. In Case 7, in which the anterior segment of the upper lobe showed typical tuberculosilicosis, the segmental bronchus was compressed by a node which was actually invading its wall. Sections of this node showed a silicotic, but not a tuberculous reaction, suggesting that the cause for a massive lesion was there before the tuberculosis. If tuberculosis is a late invader, this explains why some patients with a massive lesion show positive cultures of sputum only after a long period of observation and many examinations.

A point of great interest is the long latent period which may exist between the period of exposure to dust and the development of symptoms and signs of obstructive pneumonitis; in one of our patients exposure had ceased thirty-eight years before and in another thirty-five years previously. It is well known that diffuse silicosis of the usual type develops only over a number of years. In the case of hard rock gold miners it commonly requires an exposure period of nine or ten years. How much of this period is required to receive sufficient dosage of silica to initiate a reaction, and, this threshold dose having been received, how much of the period is required for the development of the specific tissue reaction which constitutes the disease called silicosis, is not known. Presumably, the smaller the dosage, the longer the time required to develop a significant reaction. It may be that these patients have developed the fibrotic changes in lymph nodes slowly over a period of years and then have for years had some degree of narrowing of a bronchus without serious impairment of function until a respiratory infection has led to edema

of the bronchial mucosa, increased obstruction, local pneumonitis, lymphadenitis, further pressure on the bronchus, failure of the infection to clear completely, going on to the whole picture of obstructive pneumonitis. In other words, these patients may have been vulnerable for years, but only got into difficulties following an infection which initiated a vicious circle. The history of Case 8 strongly suggests this explanation. The patient developed pneumonic attacks with increasing frequency after his first chest infection. He developed an area of pneumonitis in the apicoposterior segment of the left upper lobe which cleared slowly and then recurred in the same area just in the manner which is so commonly seen in bronchiogenic carcinoma.

Even though the picture found at operation in these cases is fairly characteristic it is difficult, and at times impossible, to be sure in any given case that there is no coexisting carcinoma. There is statistical evidence that there is no relationship between silicosis and bronchiogenic carcinoma. Whether that is true or not, patients who have frank silicosis, or like this group of patients have a type of silicotic reaction which is not visible in the roentgenogram, may develop bronchiogenic carcinoma, and we have seen examples of both types. In the patient who is known to be a silicotic, and may already have massive densities due to silicosis, the appearance of another density is likely to be attributed to silicosis, until the carcinoma has progressed to the stage of inoperability. When the patient who is not known to be a silicotic, but has the occult form of silicosis described in this paper, develops a density, it may be due to chronic obstructive pneumonitis, or tuberculosis, or to carcinoma. At operation these conditions are difficult to distinguish even with the help of frozen sections, because in each case here is a firm thickening of a bronchus with consolidation of the affected segment of lung and firm enlarged nodes. If the carcinoma is recognized it may be considered inoperable because of the presence of enlarged nodes, which are actually enlarged by a silicotic reaction. If the silicosis is recognized, it is then almost impossible to be sure that there is not an associated carcinoma.

The problem of what is the proper treatment for these patients is influenced by the necessity, in most cases, for a thoracotomy to establish the diagnosis and rule out carcinoma, although, as pointed out above, this is hard to do short of resecting the lung. One of the patients in this group had no complaints and required no treatment. The other seven were more or less disabled by their disease and in each case the disability was progressive, so that treatment was indicated. Because of the structural changes in the bronchi and the irreversible nature of the process in the lung, excision appears to be the only treatment likely to cure these patients permanently of their complaints. The one patient in the group who had thoracotomy without excision will require excision, almost certainly, before he can be considered cured of his tuberculosilicosis.

In six of these patients the massive density was confined to a single lobe or segment of a lobe, and the ideal treatment would have been lobectomy or segmental resection. In six of the seven patients subjected to operation the density of the reaction about the bronchovascular tree has been such that

nothing short of a pneumonectomy was possible for technical reasons. In one patient merely lifting the edge of the lung to examine the anterior aspect of the hilus caused an adherent node to pull away from the anterior pulmonary vein tearing a large rent in the vein. In certain cases of tuberculosis with very adherent nodes, in order to accomplish a lobectomy with safety, we have practiced a preliminary isolation and control of the main pulmonary trunk and the two veins, and have then proceeded with the lobectomy and simply repaired any injuries which the vessels sustained. It is possible that this technique might be applied to a silicotic case to permit lobectomy, but in our experience to date even pneumonectomy was difficult. In these patients one probably should not contemplate or attempt resection unless the possibility of pneumonectomy has been considered and accepted.

In assessing the result of treatment it is found that the five patients treated by pneumonectomy are all well in that they have been relieved of their chief complaints, have had no recurrence of chest infection, and have regained their normal weight or more. All are short of breath on exertion, and in three this is fairly severe. One of these three is receiving compensation on the basis of total disability and has became very obese, another is the oldest man in the group and is now 72 years of age, the third is the only one in the group who shows definite evidence of diffuse silicosis in the remaining lung. The remaining two patients who had pneumonectomy are very well and can do light work. It may be pointed out that all these patients complained of shortness of breath before operation. The patient treated by lobectomy is doing well, but insufficient time has elapsed for proper assessment. The patient who had a thoracotomy without resection has shown sputum cultures positive for tubercle bacillus and may require resection. The untreated patient remains well. On reviewing the group it is concluded that the resections which were done were justified.

It is apparent that a question of compensation is involved, and that it is a difficult question because there may be an interval of many years between exposure and the development of disability. A study of the material obtained from these patients strongly suggests that the chain of events leading eventually to severe disability was initiated by the inhalation of silica-containing dust, and would not have taken place if dust had not been inhaled. If that is true, this condition should be considered an industrial disease.

A final consideration is that of a suitable name or description for this condition. It is not silicosis, within the accepted meaning of that term, and may occur in the presence of a very little silicotic reaction in lung. The significant feature is chronic silicotic lymphadenitis; the changes in lung are nonspecific. There is no short way of saying this; perhaps "chronic silicotic lymphadenitis with obstructive pneumonitis (nontuberculous or tuberculous)" is the best that one can do.

SUMMARY

Eight cases of unilateral massive densities in lung due to chronic pneumonitis are presented. The patients were all men, and in each case clinical

and radiographic evidence strongly suggested a diagnosis of bronchiogenic carcinoma.

Five patients were treated by pneumonectomy, one by lobectomy, and in another thoracotomy was done and biopsies taken. The eighth patient has not been operated upon.

A study of the excised tissue indicates that the fundamental feature of the pathology in this group of patients is complete or partial bronchial obstruction by pressure of lymph nodes which are the site of a chronic inflammatory reaction secondary to inhalation of silica-containing dust. In five patients the chronic obstructive pneumonitis was nonspecific; in three there was an added tuberculous infection.

It is suggested that this condition be called "chronic silicotic lymphadenitis with obstructive pneumonitis (nontuberculous or tuberculous)."

The patients are all alive; the six treated by excision have been relieved of their complaints.

The author wishes to express his thanks to the members of the staff of the Department of Surgical Pathology of the Toronto General Hospital, and in particular to Dr. W. Anderson and Dr. T. C. Brown for their assistance in the interpretation of the pathology and in the preparation of the microphotographs.

REFERENCES

1. Woodruff, W., and Kelley, W. O.: Case of Massive Conglomerate Tuberculosilicosis Simulating Pulmonary Neoplasm, J. THORACIC SURG. **16**: 282, 1947.
2. Bradshaw, H. H., and Chodoff, R. J.: Anthracosilicosis Simulating Pulmonary Carcinoma, Am. Rev. Tuberc. **39**: 817, 1939.
3. Gardner, L. U.: Fourth Saranac Laboratory Symposium on Silicosis: 43-45, 1939.
4. Pendergrass, E. P.: Fourth Saranac Laboratory Symposium on Silicosis: 86, 1939.
5. Gardner, L. U.: Fourth Saranac Laboratory Symposium on Silicosis: 40, 1939.
6. Riddell, A. R.: Fourth Saranac Laboratory Symposium on Silicosis: 90, 1939.
7. Gardner, L. U.: Fourth Saranac Laboratory Symposium on Silicosis: 92, 1939.
8. Paulson, D. L., and Shaw, R. R.: Chronic Atelectasis and Pneumonitis of the Middle Lobe, J. THORACIC SURG. **18**: 747, 1949.
9. McDonald, J. R., Harrington, S. W., and Clagett, O. T.: Obstructive Pneumonitis of Neoplastic Origin, J. THORACIC SURG. **18**: 97, 1949.

DISCUSSION

DR. WARRINER WOODRUFF, Saranac Lake, N. Y.—Dr. Kergin has been kind enough to mention the case that Dr. Kelley and I previously reported, and I thought it might be worth while to review that case, because it fits in so closely with the description he has given.

(Slide.) This was a 58-year-old Polish man who had had thirty years of exposure, ten years in a hard rock coal mine and twenty years in a foundry. This mass was discovered incidentally when the patient came in for other complaints five years after his last exposure. In all the cases which we reviewed we were struck with the long latent period. There was only one case in which no exposure could be proved, but that was proved by chemical analysis. You can see how well outlined this lesion is, and the shadowing throughout. We missed the diagnosis of silicosis; we thought it was a malignancy.

(Slide.) This is the pneumonectomy specimen, and you can see the lesion there. As Dr. Kergin reported, there were a large number of hard nodules. At the time of operation we did not even suspect the condition. We did not even attempt lobectomy, and I now think it could have been done by sacrificing a certain amount of the middle lobe.

(Slide.) This is the appearance of the lesion, and the next slide shows the diffuse areas of silicosis throughout the lung.

I would agree with Dr. Kergin that the diagnosis may occasionally be made clinically, if it is suspected, particularly in the bilateral case. However, the unilateral case seems to me to pose a more difficult problem, and I am not certain that I would have had the courage not to explore his Case 7 because, as Dr. Kergin states, the fact that a patient has silicosis does not mean that he cannot also have a malignancy.

As Dr. Kergin points out, exploration in these patients is a more serious problem than in the case of an ordinary tumor because the fibrosis and adhesions at the hilum render lobectomy and segmental resection difficult and hazardous. All seven reported cases of resection have been by pneumonectomy. This is a serious matter because dyspnea is frequently the presenting complaint. Pneumonectomy further increases the dyspnea. For instance, our patient, preoperatively, had a maximum breathing capacity of 84 liters with 30 per cent residual air. Following pneumonectomy and later thoracoplasty, his maximum breathing capacity dropped to 54 liters, while his residual air increased to 41 per cent following resection and 44 per cent following thoracoplasty. Although he survived the operation, he was a "respiratory cripple" and was never able to work.

DR. JOHN F. HIGGINSON, Portland, Ore.—I would merely like to corroborate and confirm some of the points Dr. Kergin made with regard to the difficulties of dissection in the hilum in these cases. We have run across only one such case, presenting as a typical right middle-lobe syndrome on roentgenologic study, with repeated episodes of infection which necessitated diagnostic thoracotomy. We were able to do a middle lobectomy with difficulty. Stony hard nodes were very intimately bound to the vessels and the bronchus in this area, and we too tore a vessel, the pulmonary artery, where the middle lobe arteries came off. We were able to repair this, fortunately, but I think it should be emphasized that dissection is very difficult. In spite of this, however, with the necessity of making a diagnosis in this type of lesion, I do not feel that a diagnostic pneumonectomy should be done, but rather a lobectomy at the most, although it is very difficult and dangerous in these instances.

DR. JAMES M. MASON, III, Birmingham, Ala.—I wish to discuss Dr. Kergin's paper from the medicolegal aspect of silicosis. I am from an industrial section where coal and ore mining are carried on extensively by several large corporations, the United States Steel Company, the Republic Steel Corporation, Woodward Iron Company, the Sloss Sheffield Steel and Iron Company, and many smaller coal companies.

These operators are repeatedly in court defending damage suits covering the disabling effects of silicosis on the coal miners. Anthracosis is common in workers in soft coal. It is very questionable whether silicosis results from working in soft coal, which is the only coal found in this locality.

The coal companies in our section have had the laws so amended that damages from silicosis are settled on the terms of the Workman's Compensation Laws, rather than on the enormous judgments sometimes awarded in damage suits.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—Dr. Kergin has given a beautiful picture of this condition in his timely discussion. In the research project we are conducting in Knoxville with the U.M.W.A. on silicosis, we are seeing an increasing number of such cases, and during the past two weeks have explored two of them. I wish only to confirm what Dr. Kergin has already stated, particularly about the difficulties of segmental or lobar resection. These nodes are like rock, and if you have never had trouble you are in for it now.

DR. ALFRED GOLDMAN, Los Angeles, Calif.—I want to congratulate Dr. Kergin on this excellent report and to emphasize the great importance of this study, because in the past I do not think many of us have felt that silicosis was a surgical condition. But his cases show that certain forms of it are surgical.

I wish to bring to your attention a patient who had not only this syndrome which Dr. Kergin has reported, with very large lymph nodes, but also whose disability from infection was continuous over a period of months. In addition to the pathology that Dr. Kergin re-

ported there was also present, associated with the large nodes, an endobronchial granulomatous occluding process. I wonder if Dr. Kergin had any endobronchial disease in his series.

DR. KERGIN (Closing).—I wish to thank the discussers for their interest in this paper. With regard to Dr. Woodruff's comment, I thought it might arouse some doubt in your minds as to why operation was not performed in that patient. Actually, I saw him first at the time of the second film, and had I seen him at the time he had the first film I certainly would have operated on him, but having had this complete investigation I felt we could temporize in that case. And I was very much influenced by the fact that this man had no symptoms and, in my experience, I cannot do local surgery in these cases. If I plan to excise the disease I must accept the possibility that it may become necessary to do a pneumonectomy, because of possible accident to a vessel. For this reason I preferred not to operate in this case, and I am glad to say he still remains well.

Taking a biopsy does not help very much in excluding carcinoma. The hard mass is similar to the hard mass in the lung with bronchial obstruction due to carcinoma. It simply shows the type of reaction. So I think even biopsies do not exclude carcinoma unless the whole lobe can be removed and examined thoroughly. As Dr. Higginson pointed out, lobectomies can be done in some cases. I think it is much more favorable when the lobe involved is the lower lobe. In the case of upper lobes lobectomy may be technically quite impossible.

Dr. Mason has a point that I am not unaware of. This particular subject is to a certain extent dynamite, so far as industry is concerned, because if we have a situation where people are developing disability thirty years after exposure to dust and that disability is due to exposure to dust, then we have a complex and difficult compensation problem. But I think, on the other hand, that this condition is related to the dust and therefore it is an industrial disease.

With respect to Dr. Goldman's question, many of these cases have shown bronchiectasis and bronchial ulceration distal to the obstruction. We have not recognized what we could call a granuloma of the bronchus.

NODULAR TUBERCULOSIS

D. O. Shields, M.D. (by invitation), John S. Chapman, Jr., M.D., (by invitation), James Carswell, Jr., M.D., and O. J. Wollenman, M.D. (by invitation)
Dallas, Texas

DURING the past few years resection of nodular tuberculosis has become rather common. There are a number of reasons for this. Pathologic study of resected tuberculosis as stressed by Medlar has shown that tuberculous nodules usually contain a caseous center often opening into a bronchus. The fibrous wall surrounding nodules may develop slowly or be incomplete. It is thought that the caseous center may later liquify and cavities appear or spreads occur.

Prolonged bed rest usually has little effect on nodular tuberculosis. At times the nodules will decrease slightly in size and become more sharply outlined, but rarely do they disappear except over prolonged periods of time. Antibiotics may hasten clearing of the slight exudation or reversible element of nodular tuberculosis, and in some cases are of value.

Since nodular tuberculosis consists of solid tissue, the relaxation of surrounding lung by collapse therapy will have no effect on resolution of the nodules and will probably not decrease the chances of reactivation.

The above is a simplification of the problem of nodular tuberculosis, but since the point of this paper is a follow-up study of this type of tuberculosis, it will not be gone into more completely.

The development of nodular tuberculosis may occur either by tuberculous pneumonia clearing and leaving nodules, by cavities becoming inspissated, or by the formation of true tuberculomas. Many cases, however, are first seen as nodular tuberculosis, and the method of development is unknown. In addition to the difference in development, the residual nodules may vary from single small nodules to many large confluent masses. It is to be expected that the reactivation rates in these various types might differ. With this in mind a follow-up study of the various types of nodular tuberculosis was undertaken. Figs. 1 through 12 show typical examples of each type.

One hundred forty cases were selected from a Veterans Administration Hospital and from private practice for study.

Forty-nine cases were classified as residual nodules left from clearing tuberculous pneumonia. Forty were classified by National Tuberculosis Association standards as inactive at the time of discharge. Two left the hospital before activity could be determined, and seven were active at the time of discharge. Only the patients discharged as inactive need be considered, and, of these forty patients, three cases reactivated.

Thirty-one cases were followed without having previous roentgenograms, so that the development of nodules could not be determined. Twenty-four cases

From the Veterans Administration Hospital, McKinney, Texas, and Southwestern Medical School of the University of Texas, Dallas.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 8-10, 1952.

were from a Veterans Administration Hospital, and many of these were discovered from routine chest roentgenograms on admission to the hospital. Six of the twenty-four had partial calcification of the nodules, and twenty-one were upper-lobe lesions. Results are shown in Table II.

Fig. 1. Fig. 2.

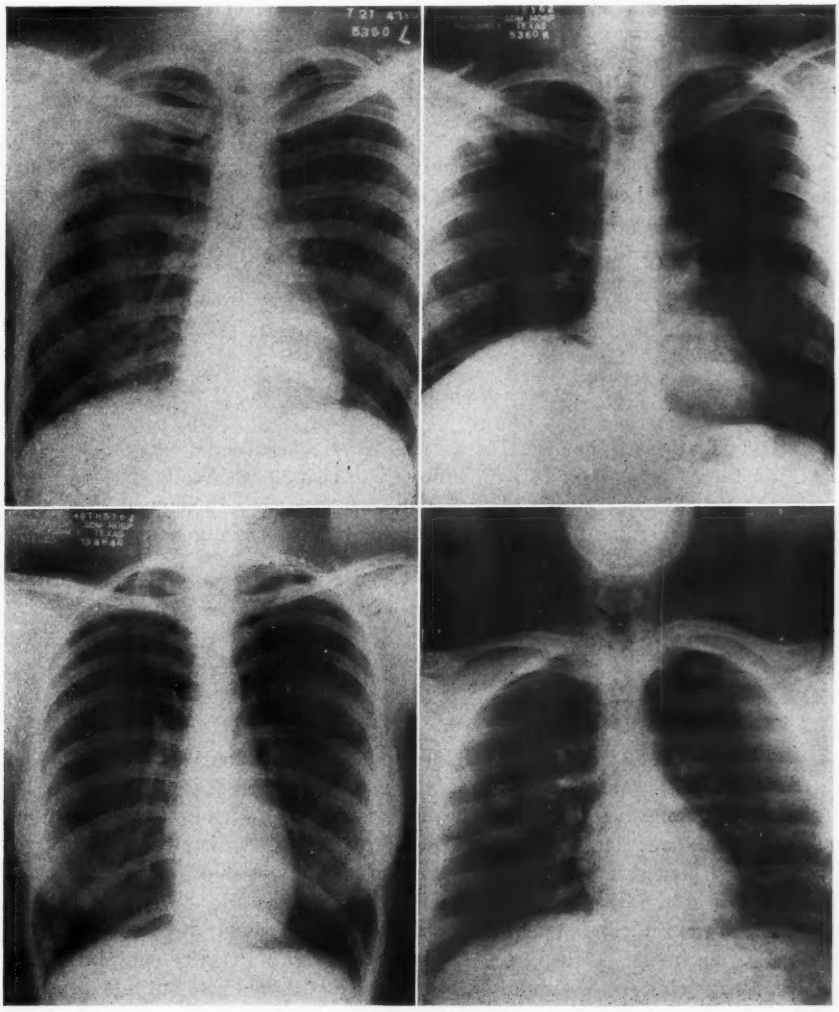


Fig. 3. Fig. 4.

Fig. 1 (Case A).—Tuberculous pneumonia, right upper lung field.

Fig. 2 (Case A).—Nodular residual which remained following clearing of pneumonia. This has remained stable fifty-two months.

Fig. 3 (Case B).—Single nodule, right first interspace. Stable twenty-four months. Development of nodule unknown.

Fig. 4 (Case C).—Partially calcified nodule, right apex. This has been stable thirty-eight months. Development of nodule unknown.

TABLE I. TUBERCULOUS PNEUMONIA DEVELOPING NODULES. FORTY-NINE CASES

Inactive (40)	Unstable 7%
Activity undetermined (2)	Unstable 50%
Active (7)	Unstable 13%
Average largest nodule	1.7 cm.
Average time followed	47 mo.

TABLE II. DEVELOPMENT OF NODULES UNKNOWN. THIRTY-ONE CASES

Inactive (31)	Unstable 6%
Average largest nodule	1.5 cm.
Average time followed	45 mo.

Ten cases of nodules developing from inspissated cavities were found by serial roentgenograms. However, only nine were inactive at the time of discharge from the hospital, and three of these showed reopening of the cavity. Two of these three cases reopened after a period of forty-eight months, and both of these patients had pneumothorax at the time of reopening.

TABLE III. INSPISSATED CAVITIES. TEN CASES

Inactive (9)	Reopened 33%
Active (1)	Reopened 100%
Average largest nodule	1.8 cm.
Average time followed	49 mo.

Only four cases that were considered to be true tuberculomas were found. All of these developed as soft infiltrations which gradually became more sharply outlined but remained the same size. No cases developed from small nodules which became larger as determined by serial roentgenograms. Results are shown in Table IV.

TABLE IV. TRUE TUBERCULOMAS. FOUR CASES

Inactive (3)	Unstable 0%
Active (1)	Unstable 100%
Average largest nodule	2.5 cm.
Average time followed	29 mo.

The last group is made up of cases which vary somewhat in their method of development. Most of the Veterans Administration Hospital patients had initial roentgengrams showing multiple large nodules with little exudation and with insignificant decrease in the size of the nodules with prolonged bed rest with or without streptomycin and para-aminosalicylic acid. Often increase in the extent of the disease occurred merely by new nodules forming. In other words, it was the type of nodular tuberculosis that slowly browses downward through the lungs. However, the cases from private practice contained many with a large element of exudation that cleared with bed rest but left a residual

TABLE V. MULTIPLE NODULES WITH FIBROSIS. FORTY-SIX CASES

Inactive (36)	Unstable 47%
Activity undetermined (1)	Unstable 100%
Active (9)	Unstable 77%
Average largest nodule	1.5 cm.
Average time followed	53 mo.

of many nodules with or without a residual of linear fibrosis. Both groups were left with a multiple nodular residual. No cases with bilateral far-advanced tuberculosis in which surgery could not have been considered are included in this or the other groups. Results are shown in Table V.

Fig. 5. Fig. 6.

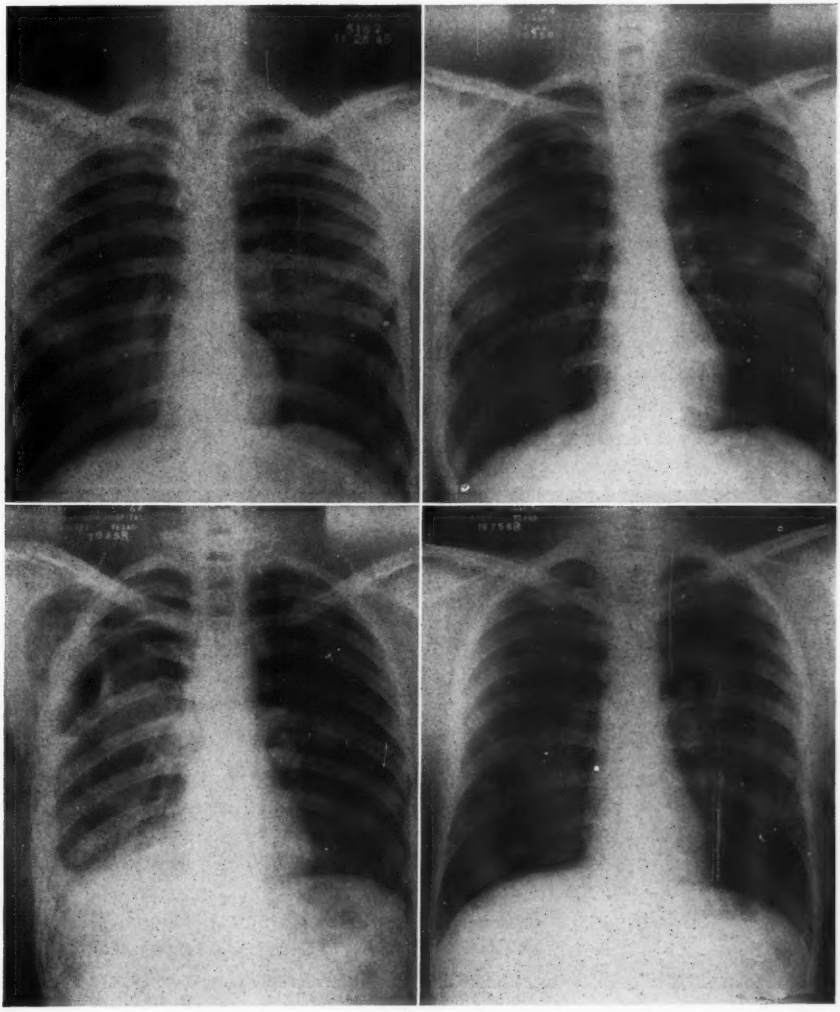


Fig. 7. Fig. 8.

Fig. 5 (Case D).—Large cavity with fluid level left middle lung field.

Fig. 6 (Case D).—Cavity became inspissated under pneumothorax. The nodule in the right middle lung field is also an inspissated cavity.

Fig. 7 (Case D).—Inspissated cavity on right reopened and recently resected. Inspissated cavity on left has reopened.

Fig. 8 (Case E).—Multiple nodular disease, right upper lobe. Disease still unstable after two years bed rest.

DISCUSSION

With the exception of the small group (4 cases) of "true tuberculomas," all groups were followed for an average period of about four years. No case

Fig. 9. Fig. 10.

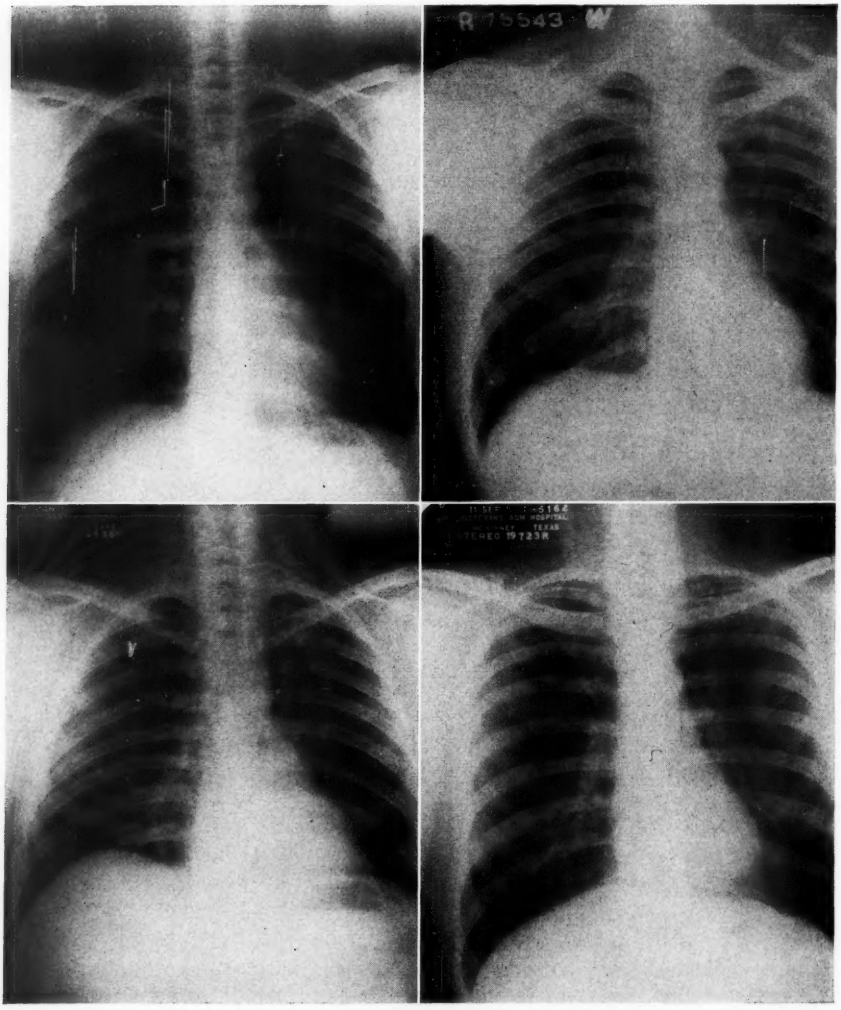


Fig. 11. Fig. 12.

Fig. 9 (Case F).—Sharply outlined nodule at left second anterior rib. Hazy infiltrate medial to this in left first interspace.

Fig. 10 (Case F).—Soft nodules in left first interspace are more plainly seen than in Fig. 9 (Case F).

Fig. 11 (Case F).—Nodules sharply outlined as compared to Fig. 10 (Case F). This was considered a true tuberculoma.

Fig. 12 (Case G).—Multiple nodular tuberculosis, left apex. No clearing on two years bed rest. Sputum cultures occasionally positive.

was included in the study if it had not been followed at least six months, and the longest follow-up was one hundred and thirty-nine months. As there is an impression that if a nodular lesion has been stable for a long period of time, it is less likely to reactivate than if it has been stable for a short period of time, an analysis of this was made. Cases followed from six to twenty-seven months were compared to those followed for longer than twenty-seven months. However, no significant difference in percentage of reactivation between the two groups were found. Groups were divided into those of nodules of 1 cm. or less and those with nodules above 1 cm., and again no significant difference in reactivation rate was found.

There were a total of 140 cases in the study and 35 cases (25 per cent) showed reactivation. If only the 119 cases which were inactive at the time of discharge from the hospital are included, there were 25 cases (21 per cent) that reactivated. A total of 76 cases in the study were from a Veterans Administration Hospital and, of these 76, 18 received collapse therapy. Eleven received a phrenic crush and of these, 4 cases (36 per cent) reactivated. Seven received pneumothorax and 4 cases (57 per cent) reactivated. It is true that only a small group received collapse therapy, but we think it supports the view that collapse therapy does not control nodular tuberculosis.

The reactivation rate was high in inspissated cavities and the multiple nodular disease groups but was relatively low in tuberculous nodules of unknown development and cases of tuberculous pneumonia where only a few nodules remained as residual.

TABLE VI. RESULTS OF CASES WITH INCREASE IN TUBERCULOSIS. THIRTY-FIVE CASES

Resected	16	
Inactive on bed rest	2	
Inactive pneumothorax	3	
Thoracoplasty	1	
Still active	11	(3 poor prognosis)
Probably active	1	
No follow-up	1	

Of 140 cases studied, 35 cases eventually became unstable either by evidence of positive sputum, increase in the lesion, or reopening of cavity as determined by x-ray study. Four of the patients that developed cavity were treated either by pneumothorax or thoracoplasty, and the disease was controlled by collapse. Eleven patients still have active disease, and 3 of these have a relatively poor prognosis. However, none of the patients have hopeless disease, and none have died. One patient has had no further follow-up after evidence of reactivation of his disease. Other results are shown in Table VI.

SURGICAL RESECTION GROUP

Thirty patients who had resection for nodular tuberculosis at least one year previously were selected for follow-up study. A total of 36 operations were done on these 30 patients, and the type of resection is shown in Table VII.

TABLE VII. EXTENT OF RESECTION

LOBECTOMY	SEGMENTAL	WEDGE OR LOCAL
14 (13 patients)	11 (9 patients)	11 (8 patients)

There were no major surgical complications in this group of patients, and no deaths occurred. The average follow-up was twenty-seven months and 2 patients have shown evidence of reactivation. One patient had a new nodule in the opposite lung two years after resection and one patient had a positive sputum culture without a change in roentgenogram fourteen months following resection. Therefore, 30 patients with nodular tuberculosis (none had cavities) which were followed an average of twenty-seven months showed a reactivation rate of 7 per cent.

TABLE VIII. RESULTS (30 PATIENTS)

SURGICAL COMPLICATIONS	ACTIVITY		AVERAGE DURATION OF FOLLOW-UP
	ACTIVE	INACTIVE	
None	2	28	27 mo.

Careful pathologic study was done on all surgical specimens. The findings were similar to those described by Medlar, and we do not think it would serve any useful purpose to describe these again.

Since the reactivation rate of the groups, (1) nodules of unknown development, and (2) nodular residuals following tuberculous pneumonia was much lower than that of the groups (3) inspissated cavities and (4) multiple nodules, a comparison of pathology in these various groups was attempted. However, no definite difference as to extent of caseation, thickness of fibrotic wall, and patency of bronchial opening could be determined in these 4 groups of nodular tuberculosis. It has been stated many times in the literature that, because of the pathologic findings in nodular tuberculosis, the lesions are extremely dangerous and the implication is that all nodular tuberculosis should be resected. We think that the pathologic findings in nodular tuberculosis are of great interest, but do not think that they should be the determining factor in selection of cases for resection. We prefer to evaluate the cases individually and resect those in which in our experience the reactivation rate has been high.

Actually, we think that most other groups who are using resection for the treatment of nodular tuberculosis are also selecting cases carefully and are not being influenced by the pathologic findings to a point where all types of nodular disease is routinely being resected.

CONCLUSION

It is dangerous to make generalizations concerning tuberculosis. However, we think the follow-up study of control cases of nodular tuberculosis and evaluation of a small group of resected cases suggest the following conclusions:

1. Inspissated cavities and multiple nodular tuberculosis have a high rate of reactivation.

2. Nodular residuals left from clearing tuberculous pneumonia and nodules of unknown development have a rather low incidence of reactivation.

3. Collapse therapy does not prevent the reactivation of nodular tuberculosis.

4. Reactivation of tuberculosis following resection of nodular tuberculosis has been low in a small series of cases, suggesting that resection may be a valuable therapeutic procedure in the treatment in this type of tuberculosis.

5. We think that resection of nodular tuberculosis should usually be restricted to inspissated cavities and multiple nodular tuberculosis.

DISCUSSION

DR. JOHN ALEXANDER, Ann Arbor, Mich.: This problem is so complex that it cannot be discussed adequately in a few minutes. Without direct reference to Dr. Shields' and Dr. Warren's series of patients (because sufficient preoperative data have not been presented) but with reference to the present wave of enthusiasm for resection in many clinics, I have observed a tendency to treat a large proportion of patients with only bed rest and chemotherapy (and without collapse therapy) for a period varying from a few weeks to many months, or even several years. Then, whether the sputum or gastric contents are negative or positive, some form of resection operation is often carried out for the roentgenographic shadows that represent the lesions that have not become absorbed. Although the present mortality rate of resection is satisfactorily low, the morbidity rate is less satisfactory and the long-term percentage of patients having consistently negative cultures of sputum or gastric contents after the cessation of chemotherapy is even less satisfactory.

Unless and until valid evidence is produced to show that small or relatively small residual roentgenographic shadows (in the presence of consistently negative cultures of pulmonary secretions two or more months after the cessation of chemotherapy) are usually or even frequently an important menace to a patient's future health, I urge that lesions suitable for collapse therapy according to the standards of a few years ago be treated by collapse therapy and that resection be reserved for those patients who are unsuitable for it or in whom it has been used and has failed.

If the frequently expressed assumption is correct that certain shadows represent caseous lesions that are virtually incapable of becoming permanently encapsulated and excluded from the bronchial tree, it is impossible for me to understand why so many thousands of patients who had lesions that had the roentgenographic appearance of small or relatively small caseous foci have lived for many years in excellent health and with negative cultures of pulmonary secretions. My plea is for a far greater discrimination in the selection of patients for resection, and for the use of suitable collapse therapy measures relatively early in the course of active tuberculous disease.

DR. B. J. RYAN, Bay Shore, N. Y.—This paper bears out Dr. Medlar's teaching concerning the hazard of the residual tuberculous caseonecrotic focus. I believe the recurrence rate in the cases just presented was from 30 to 45 per cent. If we can cut that relapse rate down from 30 to 45 per cent to the neighborhood of 5 per cent I think we have accomplished something. Dr. Medlar has repeatedly shown that obturated cavities or filled-in cavities communicate with bronchi and contain myriads of tubercle bacilli. The behavior of these lesions is unpredictable. However, those situated in anterior and inferior segments are less likely to give rise to spread than lesions in posterior or superior segments. Elimination of such foci can be accomplished with little morbidity or mortality.

It is often impossible accurately to assess the pathologic activity of a given lesion even with serial roentgenograms and careful clinical studies. Thus, adequate chemotherapy should accompany resection. I think the treatment of pulmonary tuberculosis is becoming

somewhat analogous to that of subacute bacterial endocarditis. Both can usually be controlled by long-term chemotherapy. However, surgical correction of the residual defect is necessary in some cases to prevent recurrence and provide a permanent cure.

DR. JOSEPH GORDON, Ray Brook, N. Y.—I simply would like to tell you about some of the studies we have under way at the Ray Brook State Tuberculosis Hospital. Since we were associated with the original work done at the Sun Mount Veterans Hospital, and recognizing, as Dr. Alexander has said, that this is a difficult problem, the role of chemotherapy being applied was not completely understood. We have taken a group of cases under varying courses of chemotherapy and studied them bacteriologically, and found that in a group of these cases treated from three to six months and examined carefully, they too failed to show growth of tubercle bacilli both in the guinea pig and on cultures. We have not been satisfied with this, and have selected a small group of cases with the same criteria used for surgical resection with chemotherapy, that is, a patient with a known case of tuberculosis who has shown no activity radiologically for six months and whose sputum is negative by culture for the same period of time, and have operated without preliminary drug therapy. We have resected these cases and have found that here too we have been unable to grow tubercle bacilli on culture or in the guinea pig. In addition to this, we have under way a study to determine whether or not viable organisms are present after discontinuance of drug therapy for some six months to a year. With no change in the roentgenogram and no evidence of positive sputum, resection in these patients will be done and the material examined similarly for comparison.

We have also been studying some cases by bronchography before and after resection to ascertain what changes take place in the bronchial tree postoperatively, particularly in the wedge type of operation. In some instances angiograms have also been done. In a small group of cases we have found very little evidence of distortion or damage to the lung following the wedge type of resection.

DR. FELIX HUGHES, Memphis, Tenn.—At Kennedy Veterans Hospital during the six years prior to April 1, 1952, we have performed 178 resections for pulmonary tuberculosis. I would like to make several points; (1) I think we must add a word of caution about too free an application of the principle of the excision of residual tuberculous lesions. I think one could not have a better standard to go by than the presentation of Dr. Alexander published in the Transactions of the American Review of Tuberculosis for 1951. To make an adequate choice of the proper treatment of any patient requires extensive knowledge of that patient. Our patients who were operated upon prior to Oct. 1, 1951, are reported in our paper presented here by title, and the follow-up shows that 18 per cent of these patients that have been resected have had a recurrence of disease. I want to point out, however, that many of these cases were operated upon early in our resection experience for tuberculosis, that we were using only streptomycin, that we were doing many segmental resections, and that we were doing many patients with acute or progressive disease. I think it is very important to point out that these cases must be carefully selected to get good results.

Another point I would like to make is that our results between April, 1950, and April, 1952, are greatly improved over our results prior to that time. Before that time we were using streptomycin alone. Since that time we have used at the time of resection streptomycin and PAS. We have given PAS daily to those patients who can tolerate it. I do not think there is any doubt that the combination of the two gives better results than streptomycin alone. Our results now are beginning to confirm those shown at Sun Mount, that streptomycin given one gram daily with PAS 12 grams daily, for long periods of time, will produce lesions that will show bacilli on smear, but the bacilli will not grow. We have seen this more often after daily streptomycin and PAS than we have seen it with patients who have had streptomycin twice a week and PAS daily. It has occurred in these patients too, but not so frequently.

During the last two years we have done 110 cases with three spreads, three temporary air leaks, and one fistula. All were successfully treated by ward procedures.

Another point about the treatment of late recurrences; the recurrences that occurred after resection were more easily treated than other lesions, and we have obtained better results. I would like to mention resistant cases. I do not think a patient who has bacilli that are resistant to streptomycin should be denied a resection if, from clinical examination, he is a good risk patient. We find that many of these patients have bacilli that are sensitive after the residual cavity with the resistant bacilli has been resected. We have used both the drugs in resistant and sensitive cases, regardless of the sensitivity results. I think resection now is the preferable type of treatment for residual tuberculous lesions in selected cases. The Kennedy Veterans Administration Thoracic Surgery Section Staff has now done 285 subtotal pulmonary resections for benign lesions with no deaths. This is in addition to the previously reported Army series of 236 lobectomies with one death.

DR. CHAPMAN (Closing): The original purpose of this study was to try to find out what was good to resect and what was good to leave. Our main conclusions run about as follows: nodules that developed from exudative disease, from bronchopneumonia, or from pneumonic disease, regardless of their number or their size, seem on the whole to be relatively benign and not to require resection. Nodules which developed particularly in the apical posterior regions from causes that are unknown—where we have not visualized in past films the origin of these nodules, but where we simply know they are nodules from the time they are seen, there those nodules are multiple, again regardless of their size—have an extremely bad prognosis. That is really not saying anything more than giving in a statistical fashion what everybody has for a long time thought. The type of thing that we are calling nodular disease is the same thing that Dr. Amberson in times past has referred to as indolent tuberculosis which browses downward slowly. That is what we are talking about as representing the type with the highest reactivation rate.

In answer to Dr. Alexander's comment, I would simply say that a large number of these patients either had phrenics or pneumothorax or prolonged bed rest, or a number of other things, for possibly two to three years, with stability following. And still the patients broke down again. We would regard this type of lesions as ones which fundamentally are not compressible. If they were compressible, collapse would seem to be a reasonable procedure. But these are hard; they are shotty and like marbles, and I cannot quite see how a collapse measure which depends largely upon the elastic retraction of the lung itself, would have much effect on that kind of firm, non-resilient structure.

For that reason, soon after we began to learn from Dr. Medlar what these cases were like, we began resecting them. On the whole, I think from the figures we have, that is, around 45 to 50 per cent recurrence rate or disability rate in the original group of fibronodulars, with about a 6 per cent reactivation subsequent to resection in this group, the management by resection seems preferable. It is still not a good group and I suspect, of course, that in the future as we follow these patients for longer than thirty months, we may still have our regrets.

THE COMPLICATIONS AND THE RESULTS OF TREATMENT OF BRONCHOPLEURAL FISTULA FOLLOWING RESECTION FOR TUBERCULOSIS

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OTEEN, N. C.

IN SPITE of the development of chemotherapeutic agents and refinements in surgical technique, postoperative bronchopleural fistula continues to be a frequent complication of resection for tuberculosis.

A few studies concerned with the pathogenesis of bronchopleural fistula have been made. Reports dealing with the treatment and subsequent course of a group of such patients, however, are rare. This paper will deal with the complications encountered in a series of thirty patients who developed post-resection fistulas between October, 1945, and August, 1950. The methods and the results of treatment will also be discussed. A comparative summary will be made of a group of fifteen patients who developed bronchopleural fistulas following operation and were treated during the past year.

The morbidity rate of pulmonary resection increases markedly whenever a fistula develops. This increase is usually associated with pulmonary or extrapulmonary spread of the disease. Death often results from the effects of the spread. A persistent positive sputum frequently follows the fistula and persistent sinuses are the rule.

Seventeen of the thirty patients with fistulas, or 56.6 per cent, developed pulmonary spreads. One occurred on the sixteenth postoperative day, before the fistula was diagnosed. In four patients a spread developed simultaneously with the fistula. The spreads which occurred concurrently with the fistula were extensive in character. In eight patients a spread occurred after the fistula was diagnosed. These were apparent from two weeks to eight months after establishment of the fistula. The remaining four spreads followed staged collapse procedures or appeared after the fistula was thought to be closed. It should be emphasized that the bronchocutaneous sinus must be kept open until the fistula has definitely closed. If this is not done the air-containing pocket around the fistulous opening acts like the bulb of an atomizer and causes a spray of infectious secretions to be forced into the bronchial tree with each violent cough.

The results of pulmonary spreads are shown in Table I. Six patients, or 35.2 per cent, died as a result of the spread. Three, or 17.6 per cent, have progressive disease and in eight, or 46.2 per cent, the disease is stabilized. Four have a persistent fistula.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 8-10, 1952.

TABLE I. RESULTS OF PULMONARY SPREADS

NUMBER OF SPREADS	DEATHS		PROGRESSIVE DISEASE		PERSISTENT FISTULA	
	NO.	PER CENT	NO.	PER CENT	NO.	PER CENT
17	6	35.2	3	17.6	4	23.5

Extrapulmonary spreads were less common but were uniformly fatal. Three patients incurred extrapulmonary spreads, which contributed to their death. Two of these developed tuberculous meningitis and one tuberculous pericarditis. One meningitis death occurred five months after pneumonectomy and another three and one-half months after lobectomy. Tuberculous pericarditis developed in the third patient one month after a parenchymal spread occurred. The patient died ninety days after pneumonectomy.

At the present time six of the thirty patients have persistent bronchocutaneous fistulas in spite of numerous attempts to close them surgically. Two of these have a questionable prognosis and four have shown progressive improvement. A sinus has persisted in seven patients after the apparent closure of the fistula. Six of these have stable pulmonary lesions and a negative sputum. Of the nine patients (Table II) developing a fistula following lobectomy, four fistulas, or 44.4 per cent, are closed. Twelve, or 57.1 per cent, of the postpneumonectomy fistulas are closed.

TABLE II. PERCENTAGE OF CLOSURE OF BRONCHOPLEURAL FISTULAS

	NO. OF FISTULAS	FISTULA CLOSED	
		NO.	PER CENT
Lobectomy	9	4	44.4
Pneumonectomy	21	12*	57.1

*One pneumonectomy patient died of diabetic coma at home after closure of the fistula.

Nine of the thirty patients who developed fistulas died. Eight deaths were attributable to complications resulting from the fistula. One patient died from toxemia and cardiac dilatation two weeks after an early postoperative fistula. Three died from the effects of extrapulmonary tuberculosis, as previously described. Three died as a result of massive pulmonary spread, at seven months, four months, and one month after resection. One death from cor pulmonale occurred twenty months after pneumonectomy. The final death was due to diabetic coma after discharge from the hospital. A fistula which followed a pneumonectomy had been closed by means of a thoracoplasty prior to discharge.

Seven of the nine patients who succumbed were treated with streptomycin. Eight had, in addition, an open thoracotomy and two an obliterative thoracoplasty. One patient died the day of the spread before treatment could be instituted.

Twenty-one of the thirty patients (Table III) were alive an average of thirty-six months after operation. Eleven were hospitalized but two of these were undergoing rehabilitation. Four of the hospitalized patients had open fistulas and persistent cutaneous sinuses. Ten patients had been discharged,

two with open fistulas and three with small chest wall sinuses. Three patients of the series had active disease and a positive sputum. Two of these had a doubtful short-term prognosis.

TABLE III. FINAL RESULTS OF TREATMENT

	NO. OF FISTULAS	PATIENTS LIVING	PATIENTS DEAD	IN HOSPITAL	DISCHARGED	FISTULA CLOSED*	PERSISTENT SINUS
Lobectomy	9	6	3	3	3	4	1
Pneumonectomy	21	15	6	8	7	12	6
Total	30	21	9	11	10	16	7

*Seven patients with persistent sinus are included in the closed fistula group.

Early diagnosis is of paramount importance in the treatment of bronchopleural fistula. The diagnosis was made an average of fifty-eight days following surgery in this group. Various adjuncts were used in suspected cases to help establish the diagnosis. These included bronchoscopy, instillation of methylene blue, ether, and radiopaque oils into the pleural cavity and the determination of intrapleural pressures. Gas analysis of intrapleural air was tried in two patients but did not contribute to the diagnosis.

TREATMENT

When a diagnosis of fistula was made, treatment was considered to be an emergency matter. The objectives were threefold: first, drainage of the pleural space; second, eradication of the space by the re-expansion of remaining lung tissue or obliteration by thoracoplasty; third, closure of the fistula itself.

In the majority of these patients drainage was established by means of open thoracotomy. This immediately lessened the chance of spread to uninvolved areas. As soon as the patient's condition permitted, obliteration of the potential empyema space by means of thoracoplasty was attempted. An anterior stage under local anesthesia was usually possible within a week or two after drainage. The posterior stages were then completed under local or epidural anesthesia as rapidly as the patient's condition permitted. As the infected pleural space diminished in size the patient's condition generally improved. Frequent blood transfusions were necessary during this period. An average of five or six operative procedures, including partial scapulectomies and Schede type thoracoplasties were necessary before obliteration was attained. When the space had been reduced to a narrow sinus, there were several rather dubious choices to select from. The tracts were delineated with radiopaque solutions and a determination made as to whether to attempt a plastic closure using a muscle graft, attack the bronchial defect directly, or be satisfied with a living patient who had a small draining sinus and an infrequently positive sputum. Our results with plastic closure and muscle grafts have not been encouraging. In a few patients there was apparent early success but the majority subsequently broke down and required further surgery or were treated with long-term chemotherapy.

During the past year our initial attack has been more conservative. As soon as a fistula is diagnosed a large catheter is attached to a Stedman pump and inserted through an intercostal space. This takes care of the immediate emergency and gives time to determine whether the air leak is due to a breakdown of the bronchial closure or to escape from a raw surface left as a result of segmental resection or lobar separation. Fifteen patients (Table IV) have been treated in this way. Four of these were treated with closed suction only. In three the lung was re-expanded and the temporary air leak closed in twenty-four hours. One has a definite fistula and remains on closed drainage. A secondary open thoracotomy was done on the fifth patient and a small empyema space unroofed.

TABLE IV. RESULTS IN FIFTEEN PATIENTS TREATED WITH CLOSED DRAINAGE INITIALLY

	NO.	FISTULA CLOSED	SPUTUM NEGATIVE	DEATHS
Closed drainage only	4	3	3	0
And added thoracotomy	1	0	0	0
And added thoracoplasty	5	5	5	0
And excision of remaining lobe	3	3	3	0
And secondary fistula closure	2	0	0	2
Total	15	11	11	2

Closed drainage followed by a limited thoracoplasty was done on five patients. The fistula closed, the sputum remained negative, and the wound healed in each patient.

Early open thoracotomy and excision of a remaining lobe or segment of a lobe were done in three patients with complete success. The wounds healed and the sputum became negative. Early open thoracotomy and suture of the fistula were done in two patients. Both fistulas reopened and both patients died.

The results obtained in this group of fifteen patients indicate that closed drainage, followed if necessary by a thoracoplasty or secondary resection of a remaining lobe, is a desirable method of treatment for postoperative fistula. The mortality rate in this group was 16.6 per cent. Thirteen patients are alive; eleven of them have a negative sputum and a healed wound. Two patients recently operated upon have a small residual fistula and a negative sputum.

Streptomycin and para-aminosalicylic acid were used for long periods in all of these patients with the exception of the one who died before treatment could be instituted. Conservative collapse measures such as pneumoperitoneum were used whenever spreads occurred.

SUMMARY

1. A mortality rate of 30 per cent was encountered in a group of thirty patients who developed a bronchopleural fistula after resection for pulmonary tuberculosis between October, 1945, and August, 1950. Death was usually due to the effects of pulmonary or extrapulmonary spread of the disease.

2. The mortality rate decreased to 16.6 per cent in a smaller group of fifteen patients who were treated by a modified technique during the past year. The period of observation in the later group, of course, is comparatively short.

3. Emphasis should be placed on efforts to avoid bronchopleural fistula by means of careful case selection and preoperative preparation.

4. Chemotherapeutic agents should be administered continuously for a long enough period before operation to stabilize the lesion.

DISCUSSION

DR. C. THOMAS READ, Phoenix, Ariz.—There is a period in which the patient is depleted following a fistula, especially postpneumonectomy, and a series of operations is usually necessary. I have had two experiences recently with what I would say were just fair risk patients. One had a fistula ninety days following pneumonectomy for carcinoma, and this man was a respiratory problem at that time. The other patient was a 40-year-old man who had a resection for extensive suppurative disease on the right side. He had a fistula develop in thirty days. As soon as the fistula developed, the patient was removed to the hospital from a convalescent center, a tube was inserted into the chest through an intercostal space, and the next morning each patient had a complete thoracoplasty with thoracotomy and repair of the fistula.

(Slide.) This is a 40-year-old white man who had extensive suppurative disease on the right side. I was forced to do a pneumonectomy in this situation for numerous practical reasons. You will notice on the left side the site of a thoracotomy; at the age of about 8 years, this patient had an empyema. In addition to having had drainage, a previous bronchogram disclosed basilar segmental disease on the left side. I mention this to show that this patient was only a fair risk from the standpoint of pulmonary function. In other words, he had only a partial left lung with which to breathe. This patient tolerated the procedure very well. The thoracoplasty consisted of ribs two through eight, and lysis of the pleura superiorly from beneath the first rib. He had a satisfactory postoperative recovery. The older man, who was 55 years of age, was a much worse risk than this patient from a pulmonary function standpoint, and he recovered in a like manner. Following thoracoplasty tube drainage was accomplished with water seal and suction. Both patients left the hospital in about seven days.

I realize that these operations were done on the right side, and were not done for tuberculosis. However, such a method of management might be feasible in the problem under discussion.

DR. DANIEL A. MULVIHILL, New York.—I am sure we are all very appreciative of this fine presentation of Dr. Murphy and his associates, and I wish to commend them for their very careful clinical observations, and their critical analysis of their work, which is bound to be helpful to all of us who still have to contend with this distressing complication of resection surgery.

I thought it might be of interest to you to present a parallel experience in a sister institution in the Veterans Administration, and I speak for the group at the Veterans Administration Hospital at Castle Point, N. Y. (600 beds), where Dr. Charles Lester and myself are co-consultants; Dr. Ralph Friedlander, an Associate Member, has also taken part in this work. Before showing you our results, I would stress that the operative procedures in these cases have been done over a period of five years by a changing group of personnel, and while the consultants supervise and sometimes help with the surgery, the actual pre- and postoperative care and the operative procedures have been done by a changing group of men, which has included three successive chiefs of surgery and seven junior surgeons who were in process of their training in tuberculosis surgery.

(Slide.) This is our experience for a five-year period from January, 1947, to Jan. 30, 1951, a total of one hundred twenty-eight resections, with an incidence of bronchopleural fistula of thirteen. You will note that in the last year of the series the number of bronchopleural fistulas increased greatly over the average number in the preceding four years. There are possible explanations for this, among which I would cite the fact that we were accepting poorer risk patients for the major procedures, and also operating on patients whose streptomycin sensitivity had run out.

(Slide.) This chart divides the bronchopleural fistulas into right and left. There has been a feeling that fistulas are more likely to occur on the right side; however, our experience has been a little bit to the contrary. The incidence of fistula in the entire series was 10.15 per cent; the incidence in pneumonectomy, 23.5 per cent, is about parallel to that in other reported series, as is the 6.4 per cent fistula rate in lobectomies. We have had no complications of this type in our small group of segmental reactions.

(Slide.) In thirty-four pneumonectomies we had eight fistulas, and, of these, five were complicated by spread or exacerbation of the disease. There were two deaths that occurred in pneumonectomized patients who had bronchopleural fistulas. In five cases the fistula was closed and in one case it is still open. In the lobectomy series of seventy-four cases there were five fistulas and three associated spreads or exacerbations. Two of these patients with fistula, spread, and exacerbation are dead. In two other cases the fistula was closed, and in one case it is still open.

(Slide.) This shows our total mortality figure for the 128 cases. The mortality in pneumonectomy was four deaths, 11.7 per cent; in lobectomy three deaths in seventy-four cases, 4.5 per cent. Our over-all mortality in 128 resections was 5.46 per cent.

We have been impressed with the futility of a direct attack on the fistula in an attempt to close the bronchial stump. Our approach to this complication has been similar to that of Dr. Murphy in his latest group of cases—an early closed drainage with a large tube which is inserted anteriorly, either in an intercostal space or through a small rib resection. We use a size No. 40 latex tube, and do this drainage as soon as the diagnosis of bronchopleural fistula is made. Our fistulas have occurred largely in the early postoperative period, except for one case which appeared one year after pneumonectomy, and was connected with a pocketed empyema in the residual pleural space. Following drainage we treat the patients with streptomycin and PAS, both parenterally and by injection into the residual space. When the patient is deemed in good condition, a thoracoplasty is planned and performed in stages under local anesthesia.

DR. PAUL SANGER, Charlotte, N. C.—I should like to propose two points pertinent to the care of bronchopleural fistula following resection for tuberculosis. First, I think operating upon these patients in the supine position is important, as they lie in a more physiologic position for normal breathing and there is a lessened chance of contaminating the opposite lung. Furthermore, we choose to do an anterior thoracoplasty before resecting the lung. In tuberculosis these lobes are frequently in complete symphysis with the chest wall. When there is such rigid adherence of the lung to the parietal pleura it is preferable to control the mediastinal structures before avulsing these adherently fixed lobes. If a bronchopleural fistula does develop the situation is not too grim, as already a parital thoracoplasty has been performed. Furthermore, the cavity which is under tremendous pressure can be decompressed readily by simply inserting a catheter or, better still, a small incision is made through the soft tissue of the anterior chest wall and the cavity is packed with gauze. The contamination of the contralateral lung has been eliminated by the gauze occluding the fistula. Then, with impunity, the patient can be turned to the lateral position for completion of the thoracoplasty.

After the thoracoplasty there still remains the problem of closing the fistula. This is accomplished either by an operative plastic procedure done six to twelve months later or, better, by the organization of granulation tissue, which is ideally brought about by keeping the cavity packed with gauze.

DR. LOUIS R. DAVIDSON, New York.—I should like to congratulate Dr. Murphy for truthfully telling us what he has told us. In other words, I want to congratulate him for his intellectual honesty. We recently have completed at Seaview Hospital a survey of the results obtained in cases operated upon for pulmonary tuberculosis. It was noted that between February, 1944, and February, 1952, 512 excisions on 487 patients were performed by twenty-one visiting and resident surgeons. As pointed out by Dr. Murphy and his group, we found bronchopleural fistulas to represent the greatest threat to the ultimate welfare of the patients. Eighty-two fistulas have been encountered; this is a 16.2 per cent incidence. However, you will note that in the 512 operations there were 333 pneumonectomies, which is an indication of the type of disease we are meeting with at Seaview Hospital. There were 150 lobectomies and twenty-nine segmental operations.

It was of particular interest to us in collecting these figures to note a definite difference in the incidence of fistulas on each side, as Dr. Mulvihill indicates. Fistulas on the right side are three times more numerous than those on the left. Even the lobectomies revealed the same difference. These fistulas accounted for the greatest number of deaths. In all, there were ninety-seven deaths—the over-all mortality, not from the fistulas; a mortality rate of 19.9 per cent. Of these deaths 31 were due to bronchopleural fistulas with associated empyema. In addition, there appears to be a definite age relationship to the incidence of fistulas, increasing in number with increasing years. We cautiously submit that the answer to this difficulty may be found in a modified treatment of the cartilages, a problem upon which we are engaged at the present time. No definitive statement can be made, but a very solid impression is now held that with this approach to and a consideration of the rigidity of the cartilages, the incidence of bronchopleural fistula has been very definitely decreased.

As to the methods of treating this complication, thirty of these fistulas, or 36.6 per cent, have been closed in the following manner: twelve were closed by bronchoplasty and thoracoplasty; twelve were closed by sodium hydroxide cauterization; five were controlled by completion pneumonectomy; one was controlled by removing the lingula—a completion lobectomy. Of the 31 cases treated with sodium hydroxide, 12 of the fistulas closed, four probably closed, four are still being treated, and the others are definitely unsuccessful.

It is the opinion of our group that a bronchopleural fistula should be treated early. Sodium hydroxide cauterization should be utilized before extensive empyema has manifested itself. The best results with the use of this agent have been obtained in the smaller fistulas, and especially with a granulation response that occurs after only two or four applications. Where possible, patients with frank empyema should have excisional surgery in the form of a bronchoplasty, a completion lobectomy, or completion pneumonectomy, as the case warrants. This added surgery is unfortunate, but it is perhaps the only way of salvaging patients who, in the great majority of instances, would succumb to the fistula and the resulting empyema. These operations were performed upon 487 patients; there were ninety-seven deaths; 390 patients are living; 208 patients we are now seeing in the outpatient department; forty are being followed by private doctors or other clinics; eighty-three are hospitalized, the great majority of whom have been recently operated upon. You will note that fifty-nine have been lost in the follow-up.

Of these 208 patients, 121 are on full-time activity in various types of employment. A rather unusual observation is interesting—thirty-seven of our patients had forty-one pregnancies. Four of them have had two pregnancies during or since their excisions, three during their pregnancy. Of these thirty-seven cases twenty-four had pneumonectomies, nine had lobectomies, four had segmental resection. Of the thirty-seven cases, thirty-six of these mothers continue in good health and one is dead.

I will close by quoting one sentence from the abstract of Dr. Murphy's paper: "Despite the increased morbidity following development of a fistula, the over-all results of resection have been so favorable as to result in its adoption with increased frequency."

DR. WILLIAM M. LEES, Chicago.—I think we should at least, while we are talking of the treatment of bronchopleural fistulas, also take cognizance of what causes a bronchopleural fistula. Are we perhaps operating through bronchi that have either been demon-

strated to have a tuberculous bronchitis in the past, or are we operating upon one in whom the tuberculous bronchitis still shows residual evidence of active infection, whether it be mucosal or submucosal? I mention this point because in a series of sixty-five resections, our pathologist has found an overwhelming majority showing tuberculous bronchitis at least in the submucosal layers.

The second point I wish to mention is that I think the old story of bronchial closure should be considered. The use of a noncrushing clamp, if a clamp is used, and the use of fine suture material, perhaps 00000 silk on an atraumatic needle, as I have done recently, might be the answer—I do not know. However, we have had only five bronchial fistulas, three of which we have been able to take care of with simple collapse measures. It is obvious from the discussion that most of the fistulas occur on the right side. Anatomically speaking, on the left side we know the reason why fistulas are comparatively uncommon. The bronchus recedes into the aortic window, and with one or two sutures we may close the mediastinum completely. On the right side, I think we should be extremely careful to have some form of peribronchial tissue to cover the bronchial stump and perhaps this, together with atraumatic closure, may reduce the incidence of bronchial fistulas.

DR. JAMES CARSWELL, JR., McKinney, Texas.—Our experience, although much less extensive than that of Dr. Murphy, parallels his results of early fistula as just reported. I should like to bring out the fact that in our hands the treatment of bronchopleural fistula developing late following resection for tuberculosis is a much more difficult problem than that presented by a fistula of similar degree in nontuberculous cases, particularly after pneumonectomy. Our experience with secondary closure of a wide open bronchus and plastic obliteration of remaining space in nontuberculous cases has been very satisfactory, and most unsatisfactory in tuberculous cases.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—The disastrous potentialities of bronchopleural fistula were impressed upon us early in 1947 by a case of left upper lobe lobectomy for lung abscess, where the bronchial stump eroded directly into the overlying pulmonary artery one month postoperatively. A massive hemoptysis was the first and fatal symptom. It occurred to us that the interposition of a more substantial barrier than the single layer of pleura we had heretofore used would afford greater protection and promote better healing. Since that time we have had only one significant bronchopleural fistula in our tuberculous resections. This series includes only cases where the disease was active at the time of resection, and does not include any so-called tuberculomas, wedge resections, or cases in which there was inadequate follow-up, hence we think the figure is probably significant.

On the grounds that prophylaxis is the best treatment of bronchopleural fistula, I would again like to raise the ghost that has not been laid of bronchial closure, and mention the technique which we think may be responsible for the low incidence of fistula. Essentially it is this, and there is only one small difference in it; the bronchus is divided proximal to the usual single clamp, and interrupted sutures of 00 black silk are placed across the cut end of the bronchus, these ends being left long. A free graft of pleura is taken from the chest wall and tied in a roll over the cut end of the bronchus, using the long bronchial sutures to hold it in place. This method is simplicity itself and has been, we think, most effective.

DR. FRED GRAY, Little Rock, Ark.—I think I have a right to discuss Dr. Murphy's paper because I probably left him with some of the fistulas that he has to deal with. I have nothing to say directly about the paper, but the experience we recently had with two patients might be helpful in the handling of fistulas which are recognized early. The first patient was a boy who had apparently expanded his lung well without leakage of air for about forty-eight hours, who suddenly began to leak very large amounts of air. An additional catheter was inserted in the chest, and we watched him for another three days. Then on the fifth postoperative day we took our courage in our hands and opened the chest again, finding a small laceration in a superior segmental bronchus which we had not noticed at operation. We closed this with a very few interrupted silk sutures, effected a two-rib thoracoplasty, removing the sec-

ond and third ribs, and did an apicolysis, leaving a catheter in the pleural space, with the feeling that the coaptation of the parietal pleura and the raw surface might be helpful in assuring a good sealing of the leak in the bronchus. The patient made an uneventful recovery, and has had no further evidence of fistula.

With this to encourage us, some time later we had a boy who had a very slow air leak for about seven days. We were never able to remove the tube because, although he expanded fairly well, we were afraid to do it. This time, rather than opening the pleural space, we did only an apicolysis and a modified thoracoplasty leaving the catheter in place, with the coaptation of the parietal pleura and the raw surface of the lung. This boy also had an uneventful recovery.

I would like to derive two points from this. One is, that by the conversion of an intrapleural space into an extrapleural space and the coaptation effect of the parietal pleura on the raw surface of the lung, we may effectively seal off some of these early fistulas. The second is that we were impressed by the absence of shock in these two patients who were opened through the same wound on the fifth and the seventh postoperative days. Wound healing was not delayed, and our results were satisfactory.

DR. BECKER (Closing).—I want to thank the discussers for their excellent comments and the moral support that we were given. We are envious of the people who have no fistulas to report and we sympathize greatly with those whose experience is similar to ours. We feel that awareness of a fistula is a very important thing. The diagnoses in some of these cases were not obvious and required painstaking methods to determine that the patient had a fistula. The fistulas in this series were not all early ones. One case was first detected 253 days following the operative procedure. As Dr. Murphy said, the earlier the diagnosis and the earlier the active treatment, the better chance of rehabilitating these patients.

I might also comment, since closure was brought up, that we have a series of cases which we compared with our previous series, of patients who were closed by the Overholt technique. We took the twenty previous cases and compared them with twenty we did by the Overholt method; these were pneumonectomy cases. In the previous group we had six fistulas. Using the Overholt technique we had only two fistulas. We feel that this method of closure will prove valuable in treatment of these patients.

In reply to some of the comments that were made, we have also used total thoracoplasties soon after development of the fistula. We believe that the first rib should be removed in these cases, and we also believe that one must have a patient who is fairly well stabilized for him to tolerate the procedure. We have used epidural anesthesia and it has been a great adjunct in this type of therapy. Dr. Mulvihill stated that his fistula rate increased in the last sixty-nine cases. I am afraid our experience has been very similar to this. We have taken patients who were more of a risk because of our early results, and we found that our difficulties did increase. As Dr. Sanger said, we feel that the prone position is very important, and with that position postoperative spreads have been almost minimal. We have not done concomitant thoracoplasties because we feel that if we can get along without them, we do conserve ventilatory function. We treated one case with sodium hydroxide, as Dr. Davidson has recommended. This was a case that had a fistula of fairly long duration and we were surprised when healing occurred; the patient is now discharged.

Concerning bronchial covering, we have used bronchial coverings, but we have found statistically that there was no correlation between our fistula rate and covering or not covering of the bronchi. I would like to show one slide in conclusion, which may illustrate the point that these fistulas are not always apparent.

(Slide.) This is a series of cases that we presented at the Streptomycin Conference in St. Louis in 1951, and it illustrates all the resections that were done in the Veterans Administration Hospital and those done at Oteen. Our fistula rate was higher, 12.3 per cent, but our rate of spreads and our total deaths were much lower. We feel that some of the spreads and the deaths were probably due to undiagnosed fistulas.

PLASTIC SPONGE PROSTHESIS FOLLOWING RESECTION IN PULMONARY TUBERCULOSIS

JOSEPH W. GALE, M.D., ANTHONY R. CURRERI, M.D., WILLIAM P. YOUNG, M.D. (BY INVITATION), AND HELEN A. DICKIE (BY INVITATION)
MADISON, WIS.

IT HAS been known for more than fifty years that under proper conditions and indications a foreign body implanted in the extrapleural space will definitely enhance the efficiency of collapse therapy in the treatment of pulmonary tuberculosis. Various materials have been used for this purpose, including fat, gauze, rubber sheeting, rubber bags, wire mesh, and air. None of these proved particularly satisfactory until Baer, in 1913, reported the use of paraffin. This material for years enjoyed the greatest popularity and was generally accepted as the material of choice to be used when plombage was indicated. This popularity continued until Wilson and Baker,[1] in 1946, reported their experience with the implantation of methacrylate molds in the extrapleural cavities of forty dogs. Wilson[2] later reported the results of the implantation of these molds in the extrapleural cavities of fifteen patients with pulmonary tuberculosis. Methyl methacrylate (Lucite) enjoys certain properties which many workers felt gave it a distinct advantage over paraffin. It was lighter, produced less tissue reaction, and was much easier to handle. It also possessed certain disadvantages which do not come within the scope of this paper.

As resection surgery became better developed and it was possible to remove the entire lung with a fair degree of safety, certain new problems presented themselves. It was soon realized that when an entire lung was removed for a disease such as bronchogenic carcinoma a large dead space was left, resulting in a shifting of the mediastinum to the operative side and an overdistention of the remaining lung. The overexpanded lung following pneumonectomy has been shown by Cournand and his co-workers[3, 4] to reduce the cardiopulmonary function and reserve in both adults and children. In some instances cough, pain, and dyspnea occurred following large resections. Obliteration of the dead space has been accomplished by phrenic section and thoracoplasty. One other method was to allow the cavity to fill with a serosanguineous exudate which in time became organized.

From the Thoracic Service, State of Wisconsin General Hospital, Madison.

The sponges used in this series of cases were supplied by Ivano, Inc., 123 East Twenty-First Street, Chicago, Ill. We are grateful to Mr. Corboy, the president, and to Dr. Hammon, of their research department, for their cooperation in fashioning the molds of the lobes and lungs.

These sponges can now be obtained from Clay-Adams, Inc., 141 East Twenty-Fifth Street, New York 10, N. Y.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery, Dallas, Texas, May 8-10, 1952.

It does not seem strange, then, that with the experience gained in extrapleural implantations of foreign bodies some type of prosthesis might be indicated in the pleural space after pneumonectomy. Johnson and his co-workers,[5] in 1947, reported the use of a large, hollow metal prosthesis of zirconium and also the implantation of multiple small, hollow plastic balls of Lucite. Small, Clagett, and Grindlay[6] reported on the use of a gelfoam prosthesis in 1947. This proved unsatisfactory because as time passed the gelfoam was completely absorbed. They also tried a polyethylene prosthesis which was more or less flexible and which was filled with glass wool. Each of these materials had some drawback. In 1950, Grindlay, Clagett, and Rydell[7] reported on the use of a plastic prosthesis which was made to a size roughly that of the pleural space of the removed lung. Grindlay and Waugh,[8] in 1951, reported on their experimental and clinical experience utilizing the Ivalon sponge as a framework for living tissue. These authors used the sponge in various ways unrelated to thoracic surgery, and, in addition, implanted sponges in the pleural cavities of eleven dogs following pneumonectomy. None of the dogs showed any systemic reaction. Examination of these sponges two to eighteen months after implantation revealed that there was firm fixation to the chest wall, while on the diaphragmatic surface the fibrous capsule of the sponge was covered with a smooth and glistening layer of pleura. The mediastinal surface was loosely adherent to the structures of the mediastinum. Microscopic examination of removed sponges revealed no inflammatory reaction; the sponge spaces were occupied by cellular tissue and bundles of collagen fiber around large spaces, the walls of which were lined with what resembled a single layer of vascular epithelium. Levine and associates,[9] in 1951, reported the implantation of several substances in the extrapleural space of dogs. These authors used vinyl copolymer, polystyrene, cellulose, and neoprene. Each was discarded because of some disadvantage, ranging from lack of uniform physical properties to toxic effects. Levine and co-workers, like Grindlay and Waugh, found the Ivalon sponge the most satisfactory in the experimental group. Hurst and collaborators,[10] in 1951, reported the clinical use of Ivalon in extrapleural pneumonolysis in fifteen instances. Two patients had insertion of the sponge after resection, but no mention was made as to whether it was placed intra- or extrapleurally. Charbon and Adams[11] used the Ivalon sponge in dogs to prevent overdistention of the remaining lung following resection. They concluded that the Ivalon sponge was unsatisfactory as a prosthesis since it became hard as a stone, and its adherency to the pericardium might interfere with cardiac action.

There are certain requirements which have to be met before any foreign body is placed in the tissues or utilized as a prosthesis. The material should be nonirritating, chemically inactive, stable, and produce minimum foreign body reaction. It should maintain its size and shape, be light in weight, easily removed, and, very important, it should be elastic.

Following the favorable report on Ivalon by Grindlay and Waugh, which suggested that it approached the above criteria better than any material pre-

viously used, we undertook to repeat their experiments. Furthermore, we compared the reaction produced by Ivalon with other agents commonly buried in body tissues.

Our findings corroborated those of the Mayo group. When the sponge was placed intrapleurally it became adherent to the visceral and parietal pleural surfaces in the form of a thin layer of mesothelial tissue. On palpation the sponge had an India-rubber consistency (Fig. 1).

Fig. 1.—Sponge intrapleurally—sixteen weeks postoperatively. Visceral and parietal pleurae are adherent to the sponge by mesothelial tissue. The sponge is in the center with the partially expanded lung above and the chest wall below.

On cut section an areolarlike tissue was noted between the sponge and the surrounding host tissues. This appeared less marked on the lung side, and a minimal reaction was observed about the visceral pleura. This paucity of reaction permitted a rapid development of a cleavage plane, and the visceral pleura was stripped readily from the sponge without injury to the pulmonary tissue. In the cross section (Fig. 2) it can be seen that the India-rubber consistency is due to an ingrowth of connective tissue extending into the spaces of the sponge for varying distances up to 1 cm. This invasion of the sponge by connective tissue gave the characteristic appearance of a central spongy layer and a denser peripheral one. Microscopic sections (Fig. 3) showed a very minimal or no reaction in either of the pleurae or the lung. However, vascularized fibrous tissue could be seen filling the peripheral spaces of the sponge.

The same general results were obtained when a sponge was implanted extrapleurally. Unfortunately, difficulty was encountered in developing large extrapleural spaces in dogs. Because of this it was necessary to compress the sponge in order to insert it into a relatively small bed. Sixteen weeks later when the sponge was removed it appeared flattened and somewhat dense, but it still had an India-rubber consistency (Fig. 4).

On cut section blood was readily expressed from the tiny capillaries when the sponge was gently squeezed between the fingers. The areolar tissue between the sponge and surrounding tissue was so pliable that the intercostal nerve adjacent to the sponge moved freely when traction was applied. Peeling the sponge from the adherent structures was relatively simple (Fig. 5).

These observations would indicate that the Ivalon sponge produced little or no reaction in the host tissues and that its density was modified by the de-

Fig. 2.

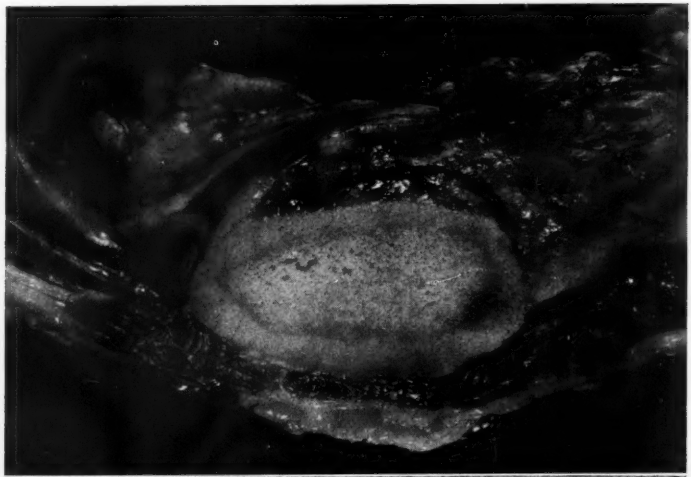

Fig. 3.

Fig. 2.—Sponge intrapleurally—cut section—sixteen weeks postoperatively. The sponge is in the center with lung above and chest wall below. Note central spongy layer and denser outer layer in the sponge. Denser area is produced by an ingrowth of fibrous tissue. No fibrotic reaction of tissues is present about the sponge.

Fig. 3.—Intrapleural sponge—microscopic section, sixteen weeks postoperatively. Lung shows no evidence of inflammation or fibrosis.

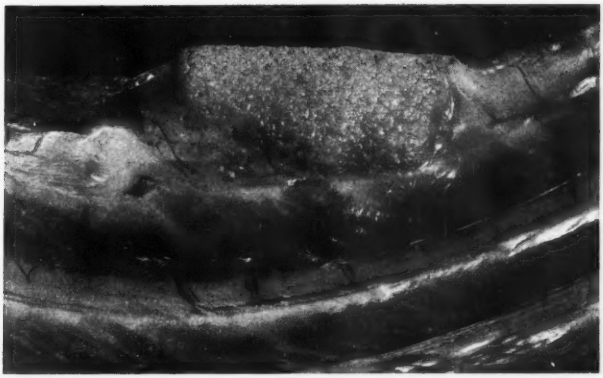

Fig. 4.—Sponge extrapleurally—sixteen weeks postoperatively. No adhesions to lung. Parietal pleura has become vascularized. Irregularity in illustration demonstrates pleura dipping into and conforming to the spaces of sponge surface.

Fig. 5.

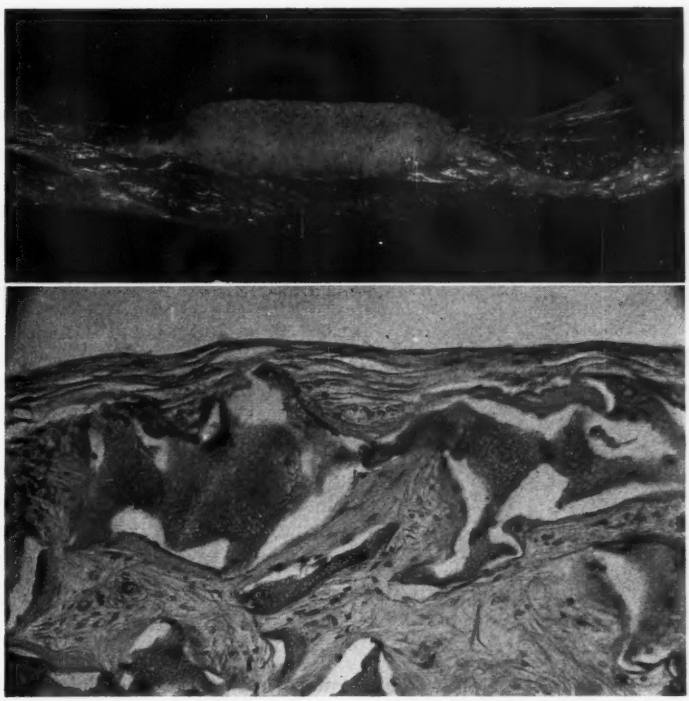

Fig. 6.

Fig. 5.—Sponge extrapleurally—cut section—sixteen weeks postoperatively. There is an absence of reaction about sponge and it was possible to move sponge on chest wall. Blood was readily expressed from capillaries within the sponge.

Fig. 6.—Extrapleural sponge—microscopic section—sixteen weeks postoperatively. The sponge is covered by parietal pleura without adhesions to lung. Capillary vessels in the pleura can be visualized.

gree of fibrous tissue growth into the sponge, the extent the sponge was compressed by its bed, and compression of the sponge while hot in the process of sterilization. Perhaps the described stonelike character of the sponge occurring in Charbon and Adams' experiments could be explained by one of these factors.

Interference with heart action with this prosthesis would be quite unlikely since the fibrous reaction is almost completely limited to the sponge, no intrapericardial adhesions are formed, and the area of pericardium involved in the tissue reaction is considerably less than one half of its entire surface.

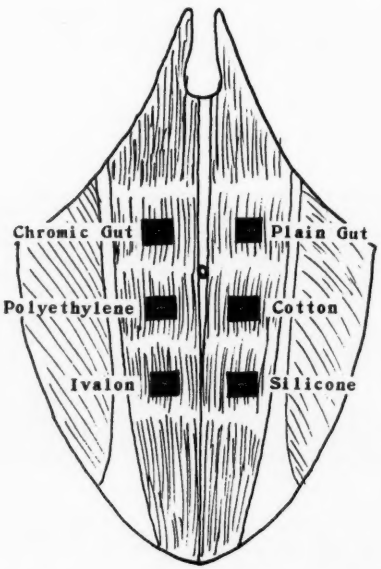

Fig. 7.—Diagram showing where implanted materials were inserted into anterior abdominal wall of a dog.

The comparative study of Ivalon sponge was made with plain and chromic catgut, polyethylene, cotton, and silicone. These substances, cut in small sheets 2 by 2 cm., were inserted through individual incisions in six different areas of both rectus sheaths of dogs (Fig. 7). The dogs were sacrificed at different times. Both recti and their sheaths were excised in their entirety, and blocks made from tissue contiguous to the implanted experimental materials.

As anticipated, the plain and chromic catgut produced a violent inflammatory reaction both grossly and microscopically in the first and second weeks (Fig. 8). By the fourth week the inflammatory exudate was replaced by fibroblasts which were beginning to lay down collagen. By the tenth week there was definite scar tissue. The sheets of gut could be seen in all the sec-

tions, but by the tenth week there was definite fragmentation. At forty-six weeks (ten and one-half months) only scar tissue was found, and all catgut had been absorbed.

Cotton, when employed in sheets, had a tendency to migrate from its bed either subcutaneously or to the peritoneum. When subcutaneous migration occurred, additional inflammatory reaction was noted. In general, there was

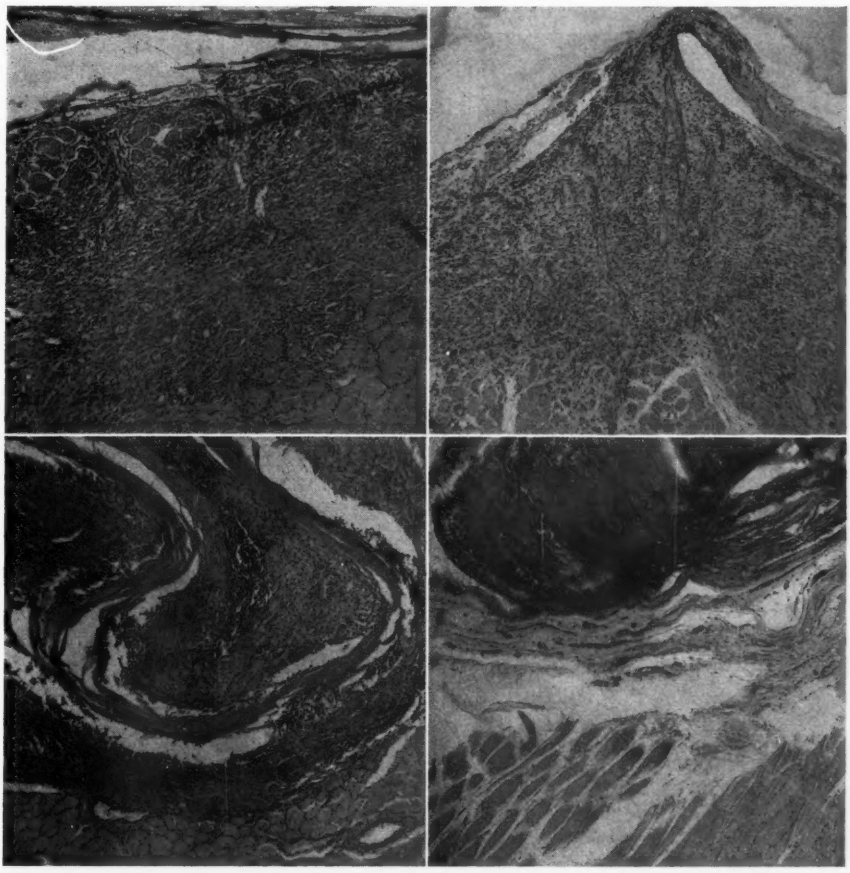

Fig. 8.—*A*, Ribbon plain catgut—one week after implantation into rectus sheath. There is marked inflammatory reaction extending well into muscle. Ribbon can be seen in extreme upper portion. *B*, Ribbon chromic catgut—one week after implantation. A similar reaction with probably less number of leukocytes. Muscle was in contact with chromic catgut membrane at the upper end. *C*, Ribbon plain catgut—ten weeks postoperatively. Most of the inflammation has subsided and is replaced by fibrosis. Can be seen undergoing fragmentation. *D*, Ribbon chromic catgut—ten weeks after implantation. Less inflammation is present than in plain gut. Fibrosis is well established, and the membrane sectioned obliquely can be visualized in the central upper portion.

much less inflammatory reaction than with the catgut, and fibroblasts were observed about the cotton fibers at the end of one week (Fig. 9). At six and ten weeks (Figs. 12 and 13) the presence of monocytes, fibroblasts, collagen, and capillaries about the cotton fibers suggested a moderate to heavy foreign body reaction.

Polyethylene manifested minimal inflammatory reaction, although in the acute phase considerable edema was seen. This edema disappeared, and from

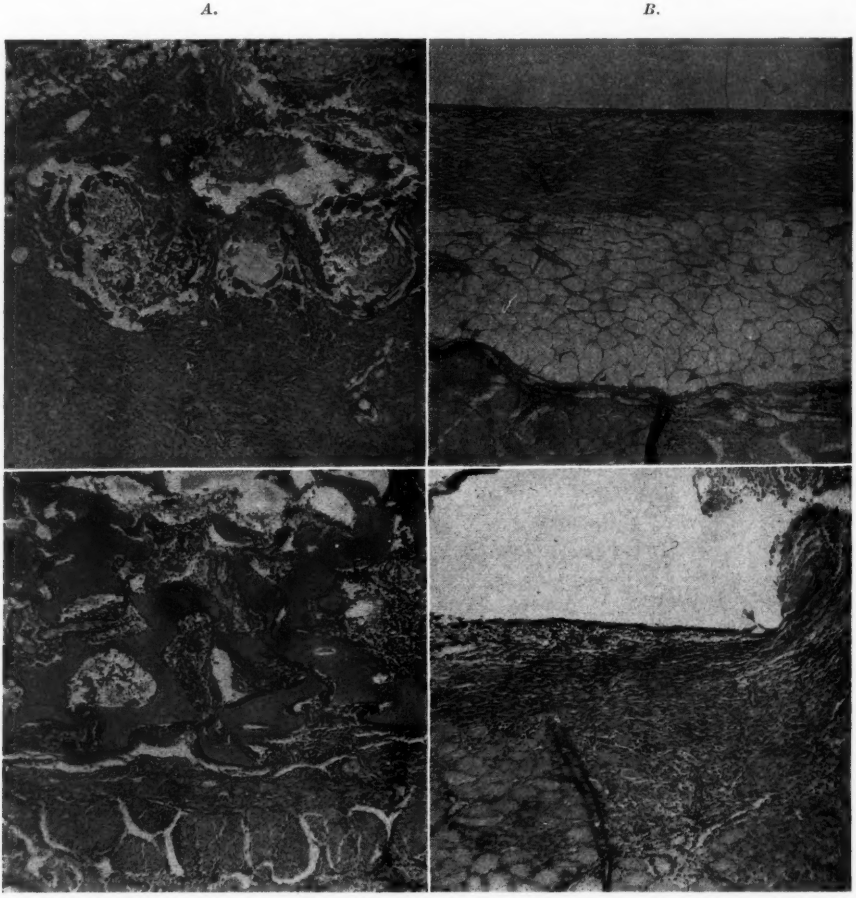

Fig. 9.—Cotton, polyethylene, Ivalon, and silicone implanted in the rectus sheath—one week postoperatively. *A*, Cotton—A moderate amount of inflammation with fibroblasts developing around cotton fibers. *B*, Polyethylene—The polyethylene was in contact with tissue at the upper end of the illustration, but fell out at time of section. Note moderate amount of fibroblastic activity. Inflammatory reaction was minimal. *C*, Ivalon—Some inflammatory cells present about the muscle fibers. Note growth of capillaries and fibroblasts into pockets of the sponge. *D*, Silicone—There is a thin layer of inflammatory reaction and fibroblasts where silicone had been in contact with muscle at the upper end.

the second to the tenth week (Figs. 10, 11, 12, and 13) a fairly thin layer of fibroblasts matured into a thin layer of dense fibrous tissue containing considerable collagen.

Ivalon in its first week exhibited only inflammatory cells between the muscle and the sponge, while fibroblasts and capillaries were growing into the pockets of the sponge. By the second week there was no evidence of inflammation, and the vascularized fibroblastic activity proceeded into the meshes of

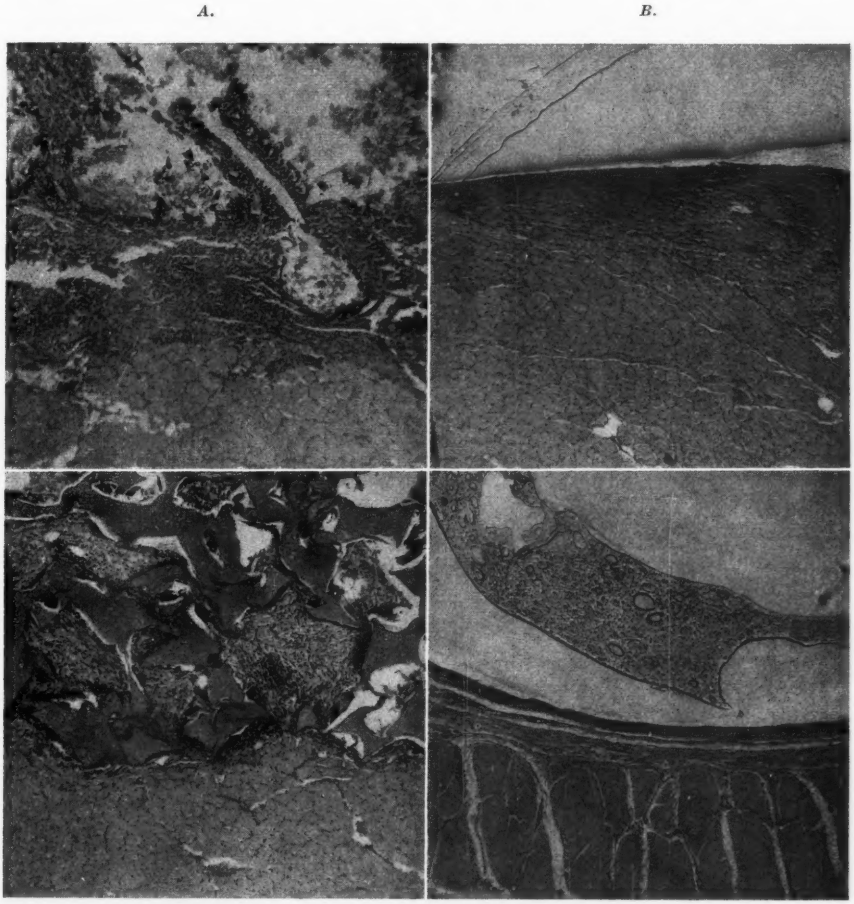

Fig. 10.—Cotton, polyethylene, Ivalon, and silicone implanted in the rectus sheath—two weeks postoperatively. *A*, Cotton—Shows profuse inflammatory reaction, fibroblasts, and some collagen. This implant migrated partially into subcutaneous tissue. *B*, Polyethylene—There is minimal fibroblastic activity seen extending into muscle at upper portion of view. The displaced film of polyethylene can be seen at the upper left corner. *C*, Ivalon—Note almost complete absence of inflammation or fibrotic reaction in muscle. The vascularized connective tissue can be seen filling the meshes of the sponge. *D*, Silicone—The silicone is present but slightly displaced. The fibrous tissue reaction can be seen as a thin rim above the muscle. No inflammatory reaction can be found.

the sponge. This ingrowth of fibroblasts continued during the ensuing weeks. At six weeks areolar tissue containing blood vessels was noted at the junction of the Ivalon sponge and the host tissues, and by the tenth week this was more pronounced.

The least reaction was visualized with silicone. Practically no inflammatory response was observed. A very narrow rim of fibrous tissue later developed about the silicone.

At forty-six weeks the gross appearance of polyethylene, cotton, Ivalon, and silicone-implanted areas showed no reaction. All implanted substances were surrounded by loose areolar tissue.

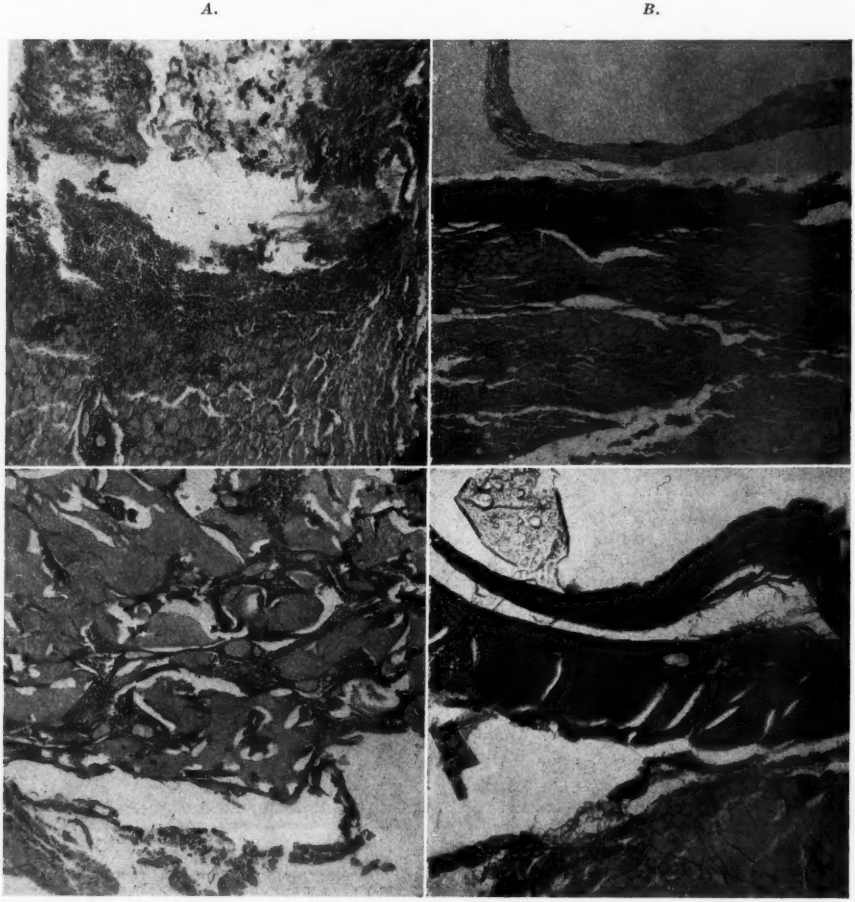

Fig. 11.—Cotton, polyethylene, Ivalon, and silicone implanted in rectus sheath—four weeks postoperatively. *A*, Cotton—Reaction is similar to the one which was seen at two weeks. *B*, Polyethylene—Presents slightly more fibroblastic activity than at two weeks. *C*, Ivalon—No inflammatory reaction. Vascularized fibrous tissue extends at least 5 mm. into the sponge. *D*, Silicone—Rim of fibrous tissue seen extending into the muscle.

The microscopic sections (Fig. 14) at ten and one-half months were difficult to cut since the tissue reaction was gone and the implanted material slipped about in the areolar tissue. Ivalon, polyethylene, cotton, and silicone demonstrated practically no reaction in the adjoining muscle tissue. Between the muscle and polyethylene, silicone, and Ivalon, areolar tissue was seen. No areolar tissue was observed with cotton. In the meshes of Ivalon dense fibrous tissue, collagen, and blood vessels were noted. Silicone showed the least reaction with a very fine layer of fibrous tissue.

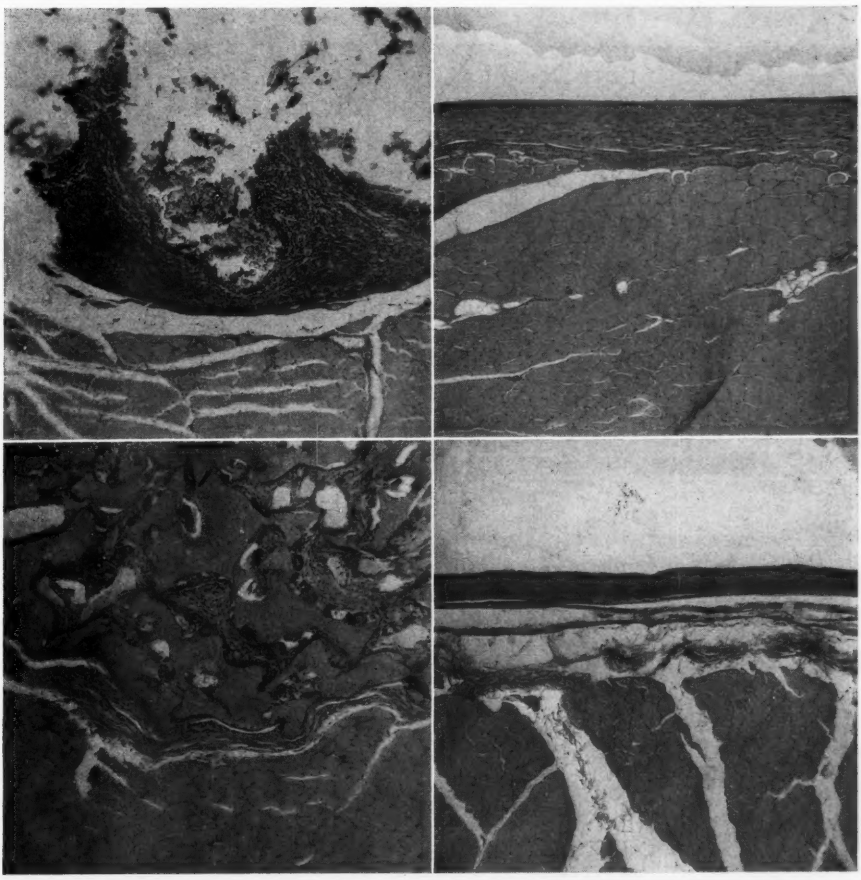

Fig. 12.—Cotton, polyethylene, Ivalon, and silicone implanted in the rectus sheath—six weeks postoperatively. *A*, Cotton, The presence of monocytes, capillaries, fibroblasts and collagen indicates foreign body reaction. *B*, Polyethylene—Thin layer of fibrous tissue in contact with muscle. *C*, Ivalon—There is areolar tissue developing at junction of Ivalon sponge and muscle. Connective tissue within sponge not that of foreign body reaction. *D*, Silicone—Thin layer of fibrous tissue present. There is some areolar tissue adjoining muscle. No fibrosis can be seen in muscle.

The polyvinyl sponge, if pure, is white in color, odorless and tasteless, and the cut surface presents the appearance of a piece of white bread. It is quite tough, and when dry becomes hard. When moistened it becomes very soft, flexible, and elastic. It is very easy to cut with scissors when soft. It is light in weight when dry, with a density of 0.05. The sponge absorbs water freely, increases in size when saturated, and on drying there is slight shrinkage.

Grindlay and Waugh found that temperatures of 120° C. had no effect on the sponge, but after being exposed to 160° C. for an hour it began to turn brown. Slight shrinkage occurred after boiling for the first time, but re-

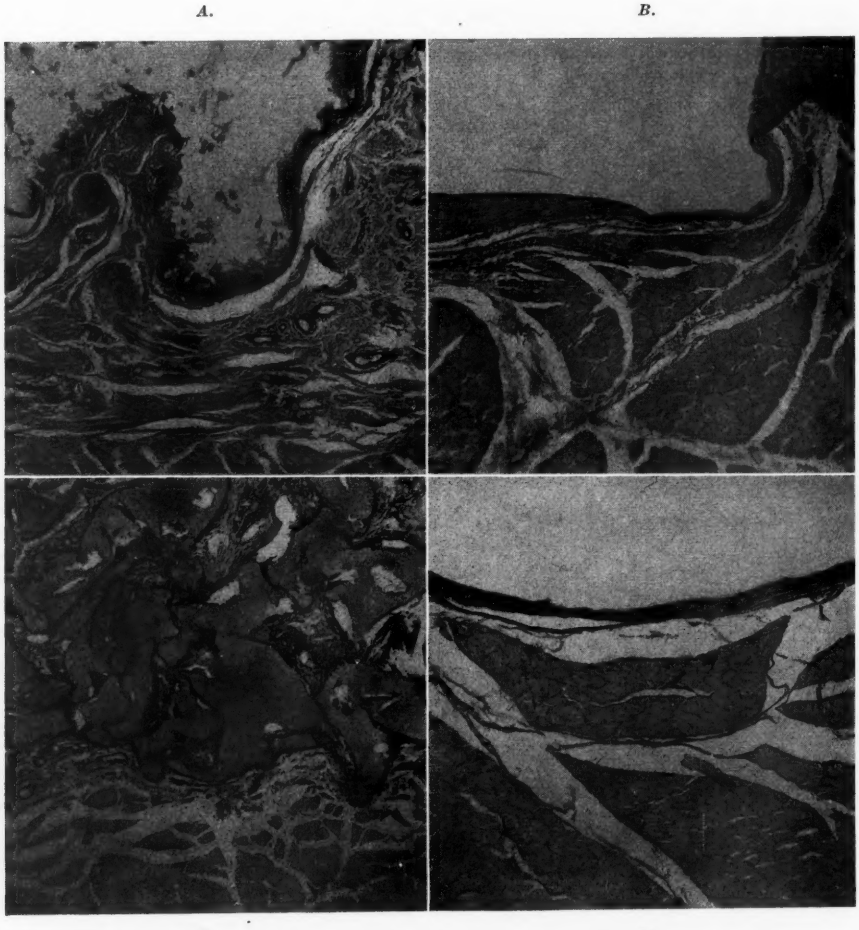

Fig. 13.—Cotton, polyethylene, Ivalon, and silicone—implanted in the rectus sheath—ten weeks postoperatively. Changes about all four implanted agents are the same as at six weeks.

peated boilings did not affect its size. They also found that if the sponge was compressed or shaped when subjected to a temperature over 95° C., the shape would be maintained when cooled.

The sponges are available in two sizes: 17.5 by 9.5 by 6.5 cm. and 33 by 19 by 14 cm. (Fig. 15). We prefer having both sizes available for use. Each sponge comes from the manufacturer packed in an airtight plastic bag which

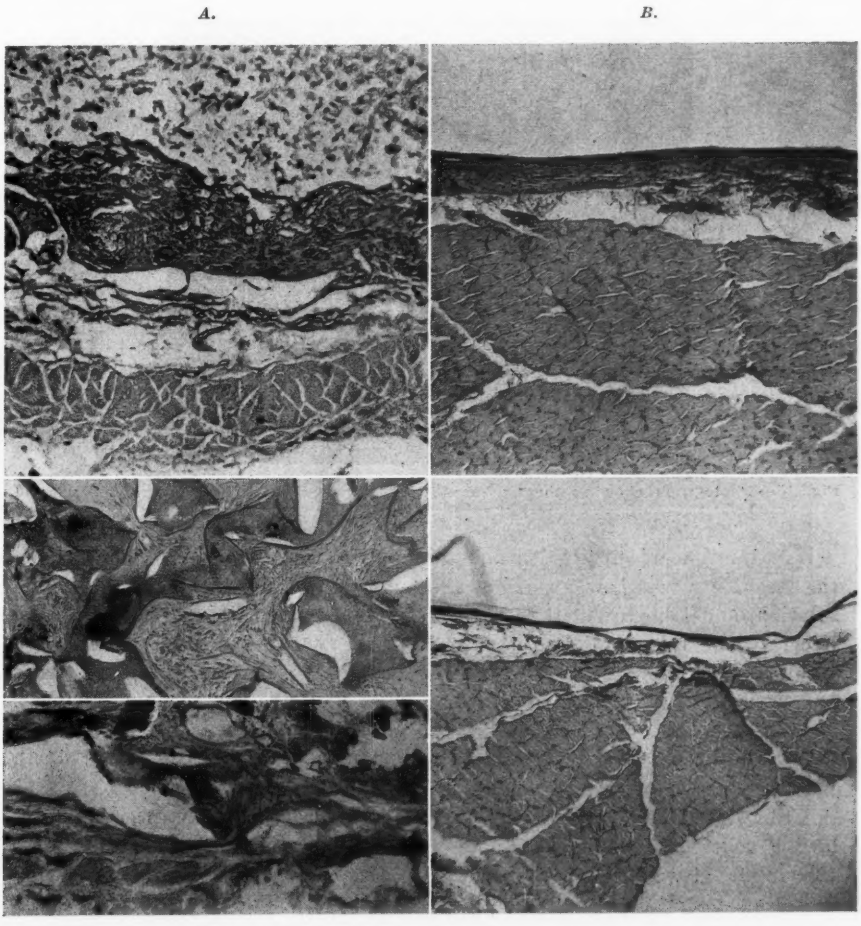

Fig. 14.—Cotton, polyethylene, Ivalon, and silicone implanted in the rectus sheath—46 weeks postoperatively. *A*, Cotton—Dense foreign body reaction about cotton. No inflammation. *B*, Polyethylene—Thin band of dense connective tissue immediately in contact with polyethylene. Areolar tissue between connective tissue and muscle. *C*, Ivalon—The extent of areolar tissue between muscle and sponge did not permit visualization of all three in a single field. No reaction is noted in the muscle. The insert above again shows vascularized connective tissue within sponge without excess collagen and no foreign body reaction. *D*, Silicone—A fine layer of connective tissue can be seen lying above the areolar connective tissue and muscle tissue where it had been in contact with silicone.

keeps it moist and soft. Upon removal from the bag the sponge should be thoroughly washed to remove the hygroscopic substance added by the manufacturer. This is easily done by squeezing the sponge several times under running water. Sterilization is simple. The sponge is placed in a clean vessel with a snug-fitting lid. It should have ample space to fit into the pan without deforming. It is boiled for thirty minutes. It may be cooled slowly, or quickly by adding cold saline solution.

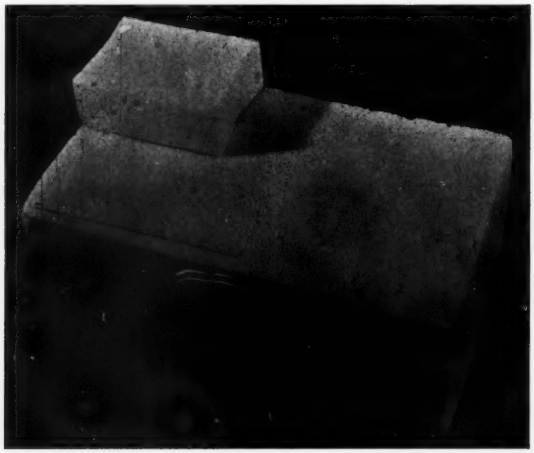

Fig. 15.—Photograph showing the two sponge sizes: small—17.5 by 9.5 by 6.5 cm., and large—33 by 19 by 14 cm.

At the time of surgery it has been our practice to remove the sponge from the sterilizing vessel, wring it out thoroughly after soaking in normal saline solution, and then to cut it to the shape desired using heavy suture scissors. Recently the manufacturers have molded the sponges into the shapes of a right and a left lung and separate lobes (Fig. 16). These are more artistic and need less sculpturing before placing into the intra- or extrapleural space. There is also less waste of time and material involved. The expense is the only deterrent to their use. Time will tell whether the added expense will be worth while.

Since April, 1951, we have utilized the Ivalon sponge as a prosthesis following lobectomy, pneumonectomy, and segmental resection in fifty cases (Table I). It was used at the time of operation in thirty-three instances, and following operation seventeen times. Twelve sponges were placed intra-

TABLE I

OPERATION	CASES	INSERTION OF SPONGE			
		AT TIME OF OPERATION		LATER	
		INTRAPLEURAL	EXTRAPLEURAL	INTRAPLEURAL	EXTRAPLEURAL
Pneumonectomy	8	1	2	4	1
Lobectomy	34	4	19	2	9
Segmental	8	1	6	0	1

pleurally and thirty-eight extrapleurally (Figs. 17, 18, and 19). We have not felt it necessary to use the sponge following lobectomy where the involved lobe is small, crenated, and atelectatic, and when the contiguous lobes have already overexpanded to occupy 85 to 90 per cent of the pleural space.

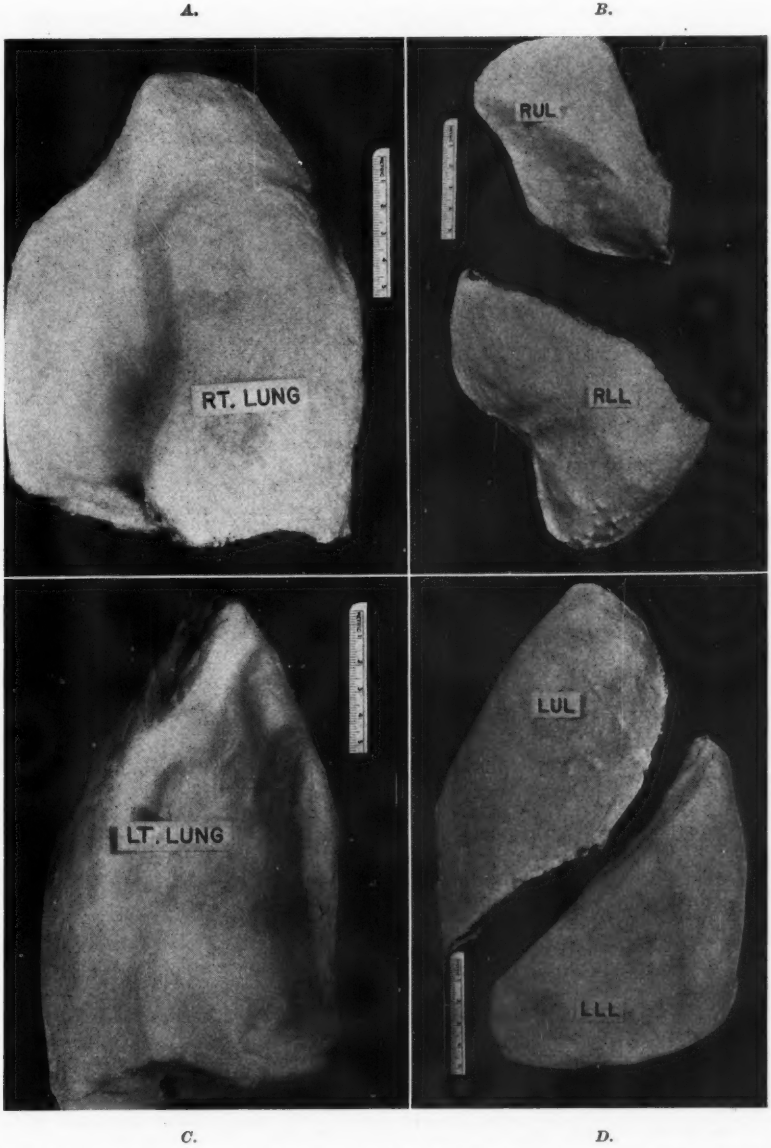

Fig. 16.—*A*, Photograph of molded right lung. *B*, Same showing right upper and lower lobes. *C*, Molded left lung. *D*, Same, showing left upper and lower lobes.

In most instances the filling has been extrapleural when done at the time of the resection. We have felt rather timid about placing a foreign body in the pleural space where we knew tuberculous lesions had been molested. In

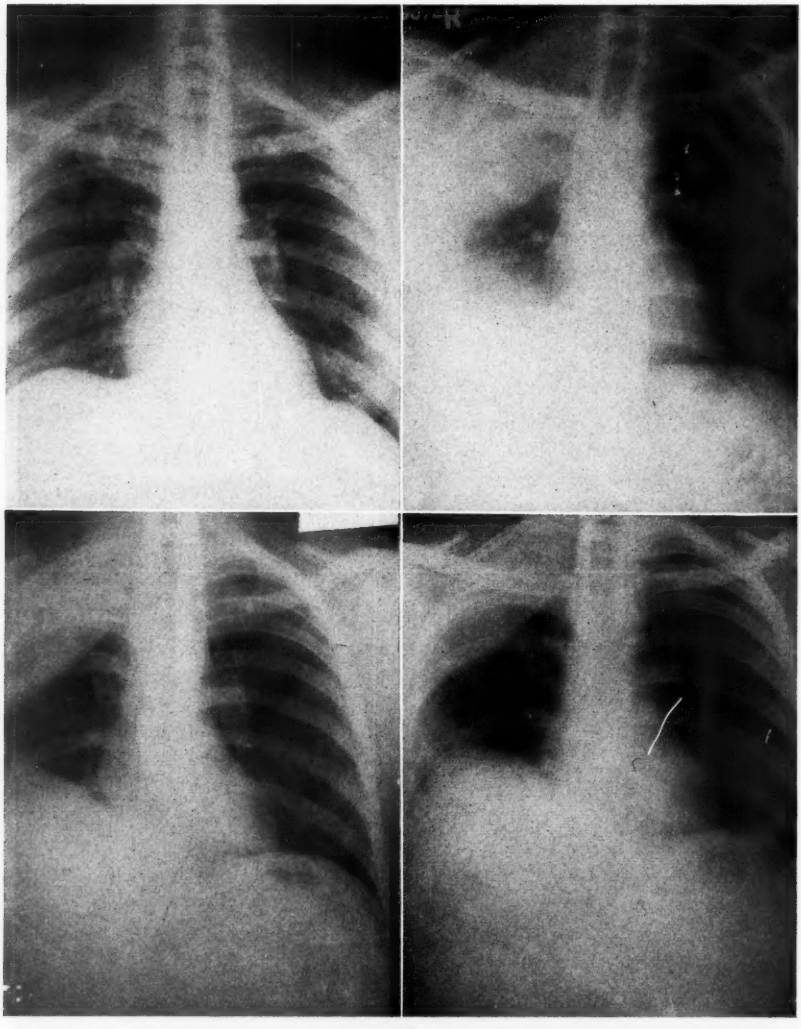

Fig. 17.—*A*, Preoperative film on patient with bilateral apical tuberculosis. *B*, Film taken four days following right upper lobectomy. The apparent large size of the sponge is due to fluid. *C*, Film taken two months later. Fluid has absorbed and lower lung field has cleared. *D*, Film taken four months after lobectomy. The size of the sponge about the same as at two months. Diaphragm high, phrenic not disturbed. Diaphragm usually returns to normal position.

these cases, following the removal of an upper lobe the parietal pleura is dissected free from the chest wall and mediastinum down to the remaining lobe. If an extrapleural dissection of the cupula was necessary to mobilize the resected lobe, the defect can be repaired easily after dissection and mobilization

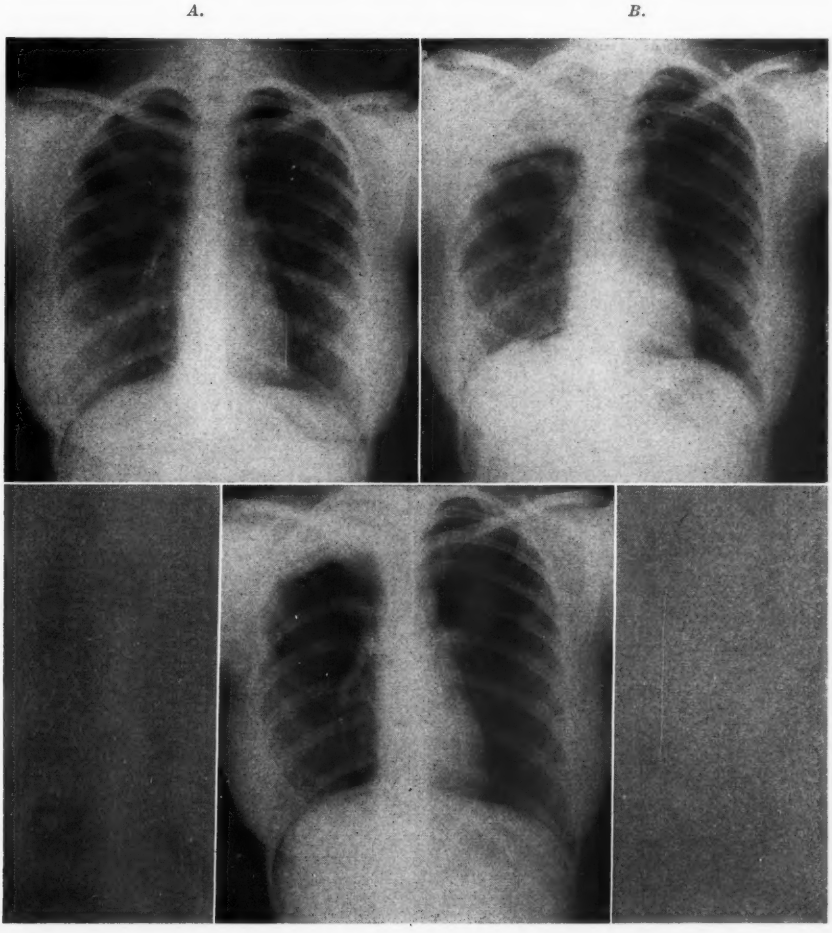

Fig. 18.—*A*, Preoperative film of patient with bilateral nodular disease, right and left upper lobes. *B*, One month later following right upper lobectomy and extrapleural insertion of sponge. *C*, Four months after insertion of sponge. There has been further reduction in size since one month. Following this it remained stable.

of the pleura. The sponge is cut to size, care being taken to make it slightly smaller than a snug fit. This seems important because the sponge not only enlarges slightly when it absorbs blood and serum, but tension and friction are lessened.

If the sponge was not inserted at the time of resection, it is a rather simple matter at a second operation to peel the parietal pleura away from the chest wall and place the pack extrapleurally. This was done in eleven cases.

The sponge has been placed intrapleurally at the time of operation in only six cases. This has been done without mishap, but our experience is not

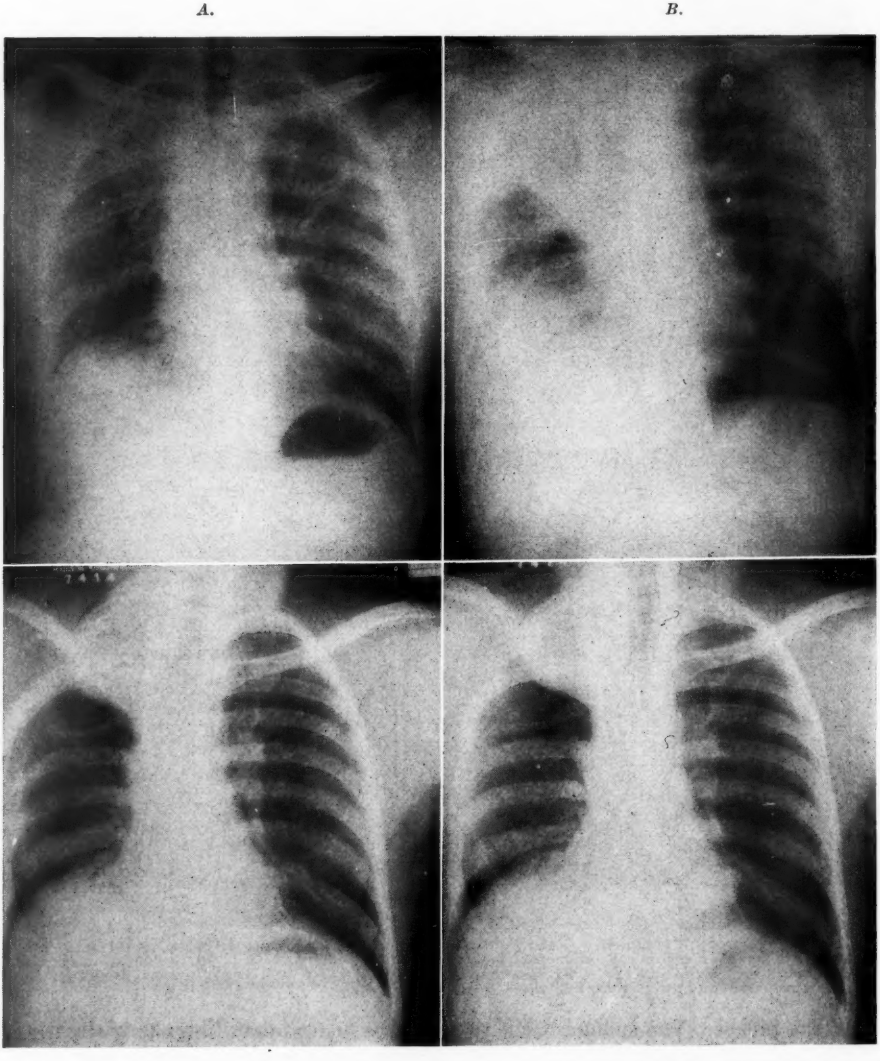

Fig. 19.—*A*, Patient ten days following right upper lobectomy. *B*, Same patient twenty-four hours after insertion of sponge at right apex. *C*, Appearance six months later. All reaction has subsided. *D*, Appearance one year later. No change noted since previous film taken at six month.

great enough to recommend it. One might postulate that following pneumonectomy particularly it might increase the hazard of bronchial fistula, which occurs too often in tuberculous cases. In pneumonectomy, where the phrenic nerve is cut at the time of operation, one cannot be sure how high the hemidiaphragm will rise and what size the sponge should be. If it were too large, pressure, friction, and complications might ensue.

In patients in poor condition, or if an extensive extrapleural dissection had been necessary for lung mobilization, and following pneumonectomy, we prefer to insert the sponge intrapleurally after waiting seven to ten days. A portion of the wound is reopened, and the pleural space is entered either through the periosteal bed of the resected rib or by resecting a portion of the fourth rib. The dead space is irrigated thoroughly with saline to remove the old clots, and the size of the cavity is determined. A large opening in the chest wall is not required since the compressible sponge can be forced through a small opening without difficulty.

As we have gained experience it appears that it makes little difference whether the sponge is placed in either the intra- or the extrapleural space at the time of the second operation. We now feel that the intrapleural technique would appear preferable since the time consumed is much less, the procedure very simple, and the results excellent.

When the sponge is inserted at the time of resection, whether intra- or extrapleurally, there is always some fluid accumulation. This is apparently not due to the foreign body reaction but appears to be in direct proportion to the extent of dissection required by the resection. The sanguineous exudate is rarely completely evacuated following lobectomy even with continuous suction. This has been found true in every instance where the pleura was opened several days following a resection. If the fluid accumulation becomes excessive following the insertion of the sponge, a thoracentesis can be done. Five patients were aspirated, but in only two of them was it repeated. Each of these necessitated three aspirations. In the remaining three cases, which were aspirated once, the procedure proved unnecessary, for in later cases the fluid was found to absorb gradually as it does following Lucite or paraffin plombage.

It has been interesting to watch the roentgenograms of these patients during the ensuing months. The sponges enlarge at first, but as the exudate and reaction subside they appear to shrink to a size even smaller than at the time of placement (Fig. 17). This change does not seem to be great enough to detract appreciably from the efficiency of the plombage. It certainly is more satisfactory and less deforming than a thoracoplasty.

In this series of fifty cases, there were no complications in forty-six. Eight sponges have been in place for one year, twenty-six for six to twelve months, and twelve for less than six months. It was necessary to remove the sponges in two instances. One patient, a diabetic, had a right upper lobectomy with an extrapleural filling at the time of the resection. He developed a wound infection due to the hemolytic Staphylococcus and an extrapleural empyema. The sponge was removed twelve days later, drainage instituted,

and later a thoracoplasty performed to obliterate the dead space. The second patient had an unexplained fever following a resection and simultaneous sponge placement. Three aspirations revealed sterile fluid. The fever subsided and the patient returned to the sanatorium with orders to continue his streptomycin and PAS therapy. This was not done, and four weeks later pus began draining from the wound. A mixed infection empyema was drained, antibiotics administered, and later the space was obliterated by a thoracoplasty. These two patients must be considered as failures. The infection in the diabetic patient was the chief offender. In the second instance, probably infection, but certainly the error in not continuing the antibiotic therapy may have contributed to the complication.

There were two postoperative deaths. One patient died eighteen hours after a segmental resection of the right upper lobe. His condition was excellent until 3 A.M. when his roommate reported his respirations were noisy and labored. He died rather promptly. At postmortem examination a tension pneumothorax was found on the opposite side. The second patient died six days after a lobectomy from a transfusion reaction. Neither of these deaths can be attributed to the sponge fill.

SUMMARY

Fifty cases of lobectomy, pneumonectomy, and segmental resection have been reported in which the polyvinyl sponge has been used as an intra- and extrapleural plombage.

By utilizing this type of prosthesis, the dead space created following resection is filled and accompanied by a minimal amount of reaction. This prevents the overexpansion of the remaining lobe or lobes on the operated side. Thoracoplasty, heretofore necessary to accomplish this, is eliminated, and the contour and functional integrity of the thoracic cage are preserved.

It is possible to complete the procedure at one sitting. If this is not advisable and a second operation is necessary to insert the sponge, it is simpler and less shocking than a thoracoplasty. We feel that in those cases where further resection is necessary on the opposite side, the patient will be a better operative risk with an intact thorax and without overdistention.

We are fully aware that as more is learned about the new drugs and their therapeutic use in tuberculosis, the amount of lung tissue which has to be sacrificed at operation will be greatly decreased. During the past year we have performed two hundred resections for tuberculosis, and 48 per cent have been of the segmental type. Many of these have consisted of the removal of such small segments that any type of fill is unnecessary. If it had not been for these changing trends our series would have been larger. Until one year ago we have found that in over four hundred resections approximately 95 per cent were lobectomy or pneumonectomy.

Any foreign material placed in the body is not ideal and will always have disadvantages. If these are minimal when compared to the beneficial effects, as demonstrated in this small group of cases, it would appear that we are justified in recommending the procedure.

REFERENCES

1. Wilson, D., and Baker, H.: Surg., Gynec. & Obst. **82:** 735, 1946.
2. Wilson, David A.: J. THORACIC SURG. **17:** 111, 1948.
3. Cournand, A., and Berry, F. B.: Ann. Surg. **116:** 532, 1942.
4. Cournand, A., Himmelstein, A., Riley, R. L., and Lester, C. W.: J. THORACIC SURG. **16:** 30, 1947.
5. Johnson, J., Kirby, C. K., Lazatin, C. S., and Cocke, J. A.: Surgery **22:** 179, 1947.
6. Small, J. T., Clagett, O. T., and Grindlay, J. H.: Proc. Staff Meet., Mayo Clin. **22:** 585, 1947.
7. Grindlay, J. H., Clagett, O. T., and Rydell, J. R.: J. THORACIC SURG. **19:** 391, 1950.
8. Grindlay, J. H., and Waugh, J. M.: Arch. Surg. **63:** 288, 1951.
9. Levine, S., Grow, J. B., Hurst, A., and Perlmutter, H.: Dis. Chest **20:** 126, 1951.
10. Hurst, A., Grow, J. B., Levine, S., and Perlmutter, H.: Dis. Chest **20:** 134, 1951.
11. Charbon, Bernard C., and Adams, William E.: J. THORACIC SURG. **23:** 341, 1952.

DISCUSSION

DR. OTTO C. BRANTIGAN, Baltimore, Md.—I would like to congratulate Dr. Gale on this beautifully presented work, and to add my brief experience with pulmonary prosthesis. Following the report by Julian Johnson on prosthesis with Lucite spheres, we did our first one in 1947, and have used the method on 33 patients. The method has been used only in the patient who had pneumonectomy for tuberculosis. The patient is allowed to recover from the pneumonectomy, and at a later date the Lucite sphere prosthesis is inserted into the intrapleural space. The prosthesis is applied under local anesthesia when the mediastinum has reached the desired position. Unfortunately three patients of the group developed infection in the pleural space about the spheres; one pure tuberculous and the other two pyogenic infections. In seeking a cause for the infection, it was found that in each instance there had been gross contamination of the pleural cavity at the time of the pneumonectomy, and the contaminated pleural space had been treated by chemotherapeutic measures sufficiently to avoid an initial empyema. There were other patients who had gross contamination at the time of pneumonectomy who have had successful prosthesis without infection. The patient who had the pure tuberculous infection about the prosthesis actually had a pleurectomy and pneumonectomy for tuberculous empyema. Prosthesis should not be used in the pleural space when gross contamination has occurred at the time of the pneumonectomy. The procedure has not been used for the patient who has had lobectomy. There have been no other complications. The first patient has had the prosthesis almost five years and he is quite well and free of trouble. There is no way to know how he will be at the end of twenty or more years.

I have found that there is a tendency for Lucite spheres to wear through where they are in contact with each other. Wilson had the foresight to insist on having the spheres sterile on the inside.

DR. EDGAR DAVIS, Washington, D. C.—I almost shudder to think what Dr. Pat O'Brien would say if he were here today. It has been recognized for a long time that occasionally, several years after the lower lobe of a lung has been removed for bronchiectasis, for example, that a patient will return with cough and expectoration of varying amounts of sputum. A repeat bronchogram on this patient frequently will reveal bronchiectasis in the upper lobe, which preoperatively had been perfectly normal. I think it is logical to assume that many times this condition is due to overdistention. For this reason I feel that some form of prosthesis would be very important, provided the proper prosthetic material can be found. Had this paper by Dr. Gale and Dr. Curreri been presented six months ago, I would certainly have been a most enthusiastic advocate of the prosthetic sponge. I have enjoyed this paper and I am delighted to know of these results. Right at this time, however, I look upon the sponge with more or less mixed feelings. I am enthusiastic in a way because of the tremendous happiness and sense of well-being, and the complete absence of structural deformity that is experienced by these patients in whom the sponge has been satisfactory. On the other hand, I regard its use with a certain amount of caution as a result of our experience with it.

Inasmuch as I had not contemplated discussing this paper, I do not have at hand my exact statistics. However, we have used sponges in about 25 patients and it is our experience in these cases that I wish to give you. In one patient who had a right lower lobectomy, the drainage tube did not function properly, and a few days later the tube was removed and decortication performed. The sponge was resterilized and replaced. Subsequently the patient developed an empyema and later died of progression of the tuberculosis. In two other patients, one of whom had had an intrapleural and the other an extrapleural pneumonectomy, it was necessary to remove the sponges. One of these patients ran an elevated temperature for from six to eight weeks, presumably from an empyema. The sponge was removed and the pleural cavity drained. Through the drainage tube and the use of antibiotics, streptokinase and streptodornase, the empyema cavity was sterilized and the tube was removed. At a later date, Lucite balls were placed in pleural cavity with a very satisfactory result. The other patient developed a discharging sinus and we waited about six weeks, hoping the sinus would close. When the sinus failed to close, the sponge was removed and the empyema cavity was drained and sterilized, much as in the case of the other patient who had had a pneumonectomy. Subsequently, Lucite balls were used with a satisfactory result. One other patient, who had a sponge prosthesis following a right lower lobectomy for suppurative bronchiectasis and multiple small lung abscesses, developed an empyema and now, six weeks later, still has the drainage tube. Possibly the sponge should not have been used in this patient, but she was a very pretty girl and anxious not to have any structural deformity. Possibly sterilization of the sponge was not as effective as it should have been.

It is readily apparent that, based upon my personal experience with the sponge, with four complications out of 25 cases, I am somewhat cautious about its use. When the sponge is satisfactory, the results are beautiful. However, an infection with a sponge is of a more serious nature than an infection without the sponge. In my considered opinion, this particular sponge may not be the ideal prosthesis for this reason. I hope, however, that this report of Dr. Gale and Dr. Curreri will stimulate the interest of other men and that the sponge or other prosthetic material will be given a further trial.

DR. ROBERT T. FOX, Chicago, Ill.—I enjoyed very much this paper by Dr. Gale and Dr. Curreri. In Chicago, we too are afraid of overexpansion of the remaining pulmonary tissue especially, as Dr. Gale has emphasized, when the resected lobe or segments are fairly large. The problems of tailoring thoracoplasty have of course been discussed before. We have been reluctant to do it at the time of resection, but consistent with our technique as described by Dr. Adams last year, we have turned again to the paraffin pack in the subscapular space, doing a limited thoracoplasty at the time of pulmonary resection in cases where we feel we want such a collapse. Unfortunately, many of these resections require major extrapleural dissection, and we have been reluctant to use prostheses here, even in the subscapular space, because of the very thin layer separating the pleural and subscapular space in such cases. But where we have a good parietal pleura we have been using such a pack, and I should like to show one case to illustrate this procedure:

(Slide.) This is a patient about 50 years of age. You see the cavity high on the left side. He was in the sanitarium just a short time and was dissatisfied with his treatment for some reason or other and left, and a pneumothorax was started on the outside.

(Slide.) Again he failed to convert to negative and continued to have cough and expectoration, and was readmitted. You see the pneumothorax here and the cavity still open, practically two centimeters in diameter in the partly collapsed upper lobe. The pneumothorax was abandoned but the lung failed to expand completely, and resection was decided upon. We did a thoracotomy and found that there was considerably more nodular disease throughout the upper lobe than would meet the eye on various roentgenograms. After resecting the upper lobe and decorticating the lower lobe, there was still evidence that the lower lobe would not fill the space satisfactorily.

(Slide.) After closing the rib cage, the second, third, and fourth ribs were resected, and a small paraffin pack of about 250 Gm. was inserted. This patient had a very ex-

cellent postoperative course. There was no paradoxical motion of the chest wall as there would be without such a prosthesis, and we feel that this is an excellent procedure in such cases. Where extrapleural dissection is necessary, we are looking forward to trying the Ivalon prosthesis, and hope that our results will be as satisfactory as Dr. Gale's.

DR. O. THERON CLAGETT, Rochester, Minn.—That surgeons are not entirely satisfied with thoracoplasty as a surgical procedure is evidenced by the variety of techniques that have been devised to accomplish the purposes of thoracoplasty without the deformity that results from it. I believe that Dr. Grindlay at the Mayo Clinic was the first to use the plastic sponge that Dr. Gale has described. About four years have elapsed since these sponges were first used in the pleural space following pneumonectomy performed in dogs. We have observed no untoward effect from the use of this sponge in the pleural space of experimental animals. The sponge prevents mediastinal shift; it causes no tissue reaction of consequence. As Dr. Gale has described, fibroblasts grow out into the meshes of the sponge, but it has been possible to remove the sponges without difficulty. Our clinical experience is much less extensive than Dr. Gale's. However, we do have one patient in which the plastic sponge has been in the pleural space for about three years and several other cases in which the sponge has been in place for shorter periods. We have had no difficulties that could be blamed on the sponge itself. We have placed the sponge both intra- and extrapleurally. It is too early for accurate evaluation of the usefulness of this material. We have had disappointments with other foreign bodies that have been used in human tissues in the past. We may be disappointed with this material in the future. However, our experience to date is most encouraging. We believe with Dr. Gale that the procedure he has described is worthy of further trial.

DR. SVEN LUNDBERG, Goteburg, Sweden.—I wish to thank you for the privilege of addressing your Association. Dr. Gale's lecture has been very interesting, and I have occupied myself with the same problems. In the surgery of pulmonary tuberculosis, the function before and after operation is more and more respected. In earlier times lung resections were done, not always accompanied by plastic operations. In many of these cases we can find a distention of the remaining lung tissue with emphysema that causes loss of function considerably greater than the diminution of function owing to the resection. In order to maintain the best function of the remaining lung tissue, I think it is necessary to reduce the size of the thoracic cavity by thoracoplasty before, at the same time, or after resection. Thoracoplasty before resection causes technical difficulties for the second operation; the simultaneous operation increases the risk; a plastic operation after resection is to be preferred, but it is not good for the patient to wait for a subsequent operation.

Within the last month I have tried in some cases to replace the resected lung or lobe with a prosthesis. However, I do not like to put foreign bodies into human tissues, and I do not perform any plombage operation in pulmonary tuberculosis. In such procedures there is some risk of injury to tuberculous tissue in the lung, and I have seen too many complications in such cases. The situation is quite different if the diseased lung or part of it has been resected. By putting a prosthesis into healthy tissue the complications will be less and probably can be eliminated if we can find a good prosthesis material. I have employed polyethelene sponge made by Ferrosan, Copenhagen. Experimental investigations with this sponge have been made by Dr. Bing and Dr. Hansen in Copenhagen, and clinical experiences have been reported by Dr. Von Rosen and Dr. Linden in Sweden and other surgeons, mainly in plombage operations. I shall not discuss the technique of plombage but will only say that the prosthesis can be put in both intrapleurally and extrapleurally. Some cases have been operated on within the last few months, and I should like to show you this sponge material.

(Slide.) The first case is one of stenosis of the main bronchus on the left side. This film was taken some days after pneumonectomy. The sponge is seen in the upper part,

and in the lower part the prosthesis with some exudate and displacement of the heart to the left. (Slide.) Two months after resection the heart is in normal position and (Slide) three months after resection the heart is still in normal position.

(Slide.) This is a case of pneumothorax with the remaining cavity. The picture was taken some days after resection and again you see displacement of the heart to the right. (Slide.) One month later there is exudate and we can see the sponge in the upper part. The heart is in normal position and (Slide) this was taken two months after resection.

(Slide.) You see in this case a great cavity in the right upper lobe. I did a six-rib plastic, but there was a remaining cavity at the apex. (Slide.) Then I performed a resection of the right upper lobe and put in a prosthesis and, as you see, below that there is exudate, but I hope that after some months the exudate will be resorbed and that we will have normal function of the lower part of the lung.

DR. GALE (Closing).—I wish to thank those who were kind enough to discuss this paper. I do not want to leave the wrong impression. Certainly one would be rather foolish to claim too much for a sponge such as this after using it in only 50 cases. It is a foreign body. Many of you who started using paraffin years ago know how tricky it was, as well as any prosthetic appliance of any kind.

I think we must continue to feel our way, and if there is any doubt as to the presence of contamination of the intra- or extrapleural space at the time of operation, please do not insert the sponge. I also feel that as time goes on and more experience is gained, we will know just how safe it is. It is not the type of procedure that can be carried out by everyone or on every case. Each patient will have to be evaluated separately.

CIRCULATORY ARREST

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CIRCULATORY arrest after local anesthesia or during operation has such a high mortality that the generally accepted principles of treatment must be revised.

ETIOLOGY

The cause of circulatory arrest is in most cases an anoxia and the best treatment is prevention. In cases with local anesthesia idiosynerasy to the drug or injection in a vessel may be the cause.

PATHOPHYSIOLOGY

During operations with the chest open the development of a circulatory arrest has been observed several times : In most cases the heart rate is rapidly slowing down to 10 to 20 beats per minute. A standstill of the heart then comes in a very short time. In very few cases the circulatory arrest is preceded by a gradual increase in the heart rate, an increasing number of extrasystoles, and finally ventricular fibrillation. The heart may also come into a ventricular fibrillation secondary to a standstill, especially during a delayed resuscitation.

Differential diagnosis between a heart standstill and a ventricular fibrillation can, in a short time, be made only by direct inspection of the heart in these catastrophic cases. If a direct writing electrocardiograph is connected, the ventricular fibrillation is demonstrated, but in standstill atypical and misleading tracings may be produced.

TREATMENT

Theoretical Considerations.—The treatment is entirely different in the two groups. Injection of Adrenalin is indicated in cases of heart standstill but contraindicated in ventricular fibrillation, where the only effective treatment is an electric shock. Therefore the logical treatment, which is now generally accepted, is: (1) opening of the chest and the pericardium for diagnosis and (2) heart massage, (3) artificial positive pressure ventilation through an endotracheal tube, (4) injection of Adrenalin into the heart in the case of a heart standstill if heart massage alone does not prove sufficient, (5) electric shock direct to the heart in case of ventricular fibrillation, and (6) continuation of heart massage and, if necessary, repetition of the measures mentioned under 4 and 5.

PRACTICAL CONSIDERATIONS

A dog's heart may be without oxygenated blood for 33 minutes and then resume regular rhythm with complete survival, provided brain perfusion with oxygenated blood is performed. If the human brain, however, is without

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oxygenated blood for more than 3 to 4 minutes, irreparable changes will occur. Therefore two requirements have to be fulfilled to permit the above mentioned scheme: (1) the chest must be opened and the heart massage instituted within 3 minutes from the time the diagnosis is made, allowing a maximum of one minute to establish the diagnosis, and (2) a defibrillator must be at hand for the electric shock. If these two requirements are not fulfilled the result will only be a temporary restitution of normal cardiac action in a decerebrated patient.

The above mentioned scheme should then be followed routinely in all cases with an open chest during operation and in most cases during an intervention in the upper abdomen. In all other cases of circulatory arrest the author follows a more conservative plan.

1. Adrenalin, 1:1000, and a syringe with a long, widebore needle is always at hand. As soon as the diagnosis is established the needle is inserted into the heart 2 fingers breadth lateral to the sternum between the fourth and fifth ribs on the left side. A widebore needle enables the surgeon to quickly aspirate blood and thereby make sure the needle is accurately placed in one of the chambers of the heart.

2. One milliliter of Adrenalin, 1:1000, is injected. If this is done quickly enough and the heart is still beating slowly, an immediate response is noted; the pulse returns and the needle, which moves with the heart beat, can then be removed.

3. If, a few seconds after the first injection, no response is noted 5 ml. Adrenalin, 1:1000, is injected. An immediate response may be noted in most instances. If not, 10 ml. Adrenalin 1:1000 is given.*

During these injections only one minute is lost. In the meantime artificial positive pressure ventilation is instituted through an intratracheal tube.

Following this scheme it has been possible to re-establish a regular cardiac beat in 4 successive cases. In two cases the recovery was complete, in the other two an immediate response to 5 ml. Adrenalin, 1:1000, was achieved, but the injections were made too late and cerebral damage had already occurred.

The value of intracardiac Adrenalin injections in cardiac resuscitation has been underestimated. Physiologists have warned us and described the bad reaction to Adrenalin of the beating heart in experimental animals. An intravenous injection of 0.5 ml. of Adrenalin 1:1000, made by mistake on a healthy 20-year-old girl was lethal.† Most surgeons use only 1 ml. of Adrenalin, 1:1000, which is too little and is often without effect.

Two such cases with circulatory arrest after local anesthesia for thoracoplasty were experienced in 1949.

*A combination of Adrenalin and 5 ml. 1 per cent procaine may be valuable. As, however, speed is the over-all important thing, the Adrenalin alone is preferred if some seconds are gained by omitting the procaine.

†On the other hand Crafoord during an intrathoracic operation by mistake injected 10 ml. Adrenalin, 1:1000, in the heart at standstill. There was an immediate response with complete recovery and no untoward effects.

The heart in standstill is anoxic, the reaction delayed, and a big initial dose is needed. No untoward effect is noted once the heart is beating again as the action of Adrenalin is too short.

CASE REPORTS

CASE 1.—July 21, 1949, a 41-year-old man had local anesthesia with paravertebral block, brachial plexus block, and infiltration in the line of incision of the subcutaneous and muscle planes (0.5 per cent procaine with tetracaine 0.5:1000). About 3 to 4 minutes after the completion of the anesthesia, the patient's pulse suddenly stopped. Artificial ventilation through an intratracheal tube and 1 ml. Adrenalin, 1:1000, in the heart had no effect. The patient died. Post-mortem examination showed right-sided pulmonary tuberculosis.

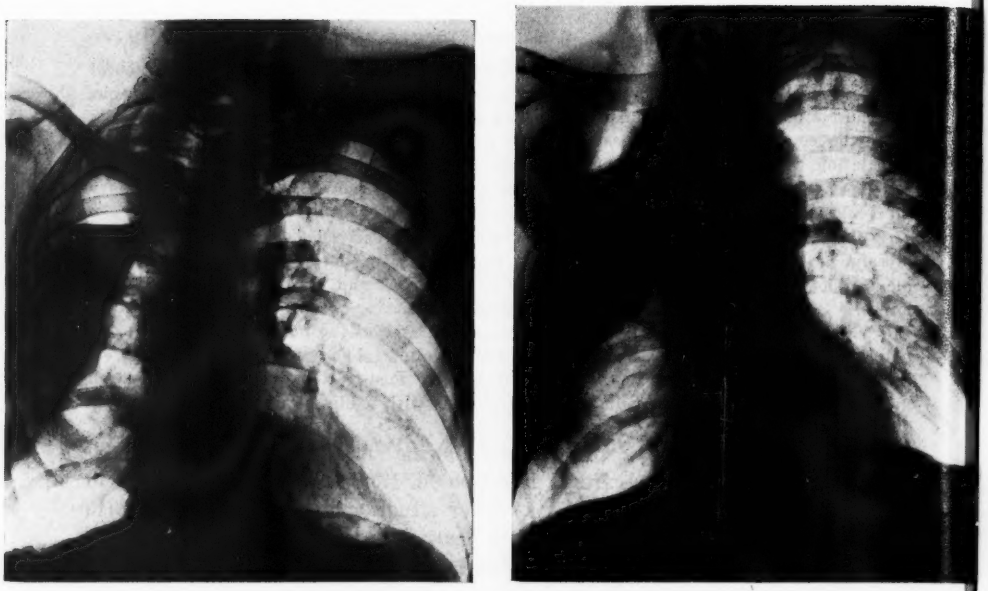
CASE 2.—November 8, 1949, a 27-year-old woman received 230 ml. of 0.5 per cent procaine with tetracaine 0.5:1000 in local anesthesia with paravertebral block, brachial plexus block, and local infiltrations in the subcutaneous and muscular planes. Fifteen minutes after completion of the local anesthesia and during performance of the incision, the pulse rate suddenly increased but after a short interval the pulse could no longer be palpated. Intracardiac injection of 1 ml. of Adrenalin, 1:1000, and artificial ventilation through an intratracheal tube was without effect. Post-mortem examination revealed nothing other than bilateral pulmonary tuberculosis with a big cavity in the right upper lobe.

With this experience of two deaths following circulatory arrest after local anesthesia treated by intracardiac injection of 1 ml. Adrenalin, 1:1000, and artificial ventilation, I decided, in a similar situation, to follow the advice to perform an immediate thoracotomy with heart massage. It is, however, easier to make the decision than to carry out the treatment if the surgeon is operating in a sanatorium which is equipped only for thoracoplasties made under local anesthesia which are performed by the surgeon himself.

CASE 3.—Two years later this case of circulatory arrest occurred after epidural anesthesia with 30 ml. Xylocain 1 per cent for a second stage thoracoplasty. The patient was a 57-year-old man with a right-sided tuberculous empyema, who 25 days earlier had undergone a first stage 5-rib thoracoplasty with apicolysis. It was clear that the generally accepted treatment with cardiac massage was impossible. The right lung was too diseased with an atelectatic upper lobe and a big empyema to take over the function alone during and after a left-sided thoracotomy for cardiac massage. A right-sided thoractomy could not be performed in 3 minutes (see roentgenogram, Fig. 1). It had been easy for a chest surgeon to open the left chest. The outcome of such a treatment with cardiac massage would, however, most probably be fatal. For that reason the following treatment was instituted: (1) Immediate intracardiac injection of 2 ml. Adrenalin, 1:1000, without response. (2) The surgeon introduced an intratracheal tube and gave artificial ventilation by intermittent blowing his own expiration air into the tube during the time the anesthesia machine was connected. (3) After 3 to 4 minutes of cardiac arrest 8 ml. of Adrenalin, 1:1000, was injected into the heart with immediate return of the pulse which could be felt for 15 minutes. (4) Then another cardiac arrest occurred. After immediate intracardiac injection of 10 ml. of Adrenalin, 1:1000, regular heart beat reappeared. One-half hour later, spontaneous breathing began and after 7 hours, the intratracheal tube could be removed. After 2 days he had recovered completely, but had no memory of these 2 days.

In all, 20 ml. of Adrenalin, 1:1000, was given intracardially, which saved this patient's life. With this experience I cannot agree with Ehrenhaft and others (1951), who make the conclusion that, "epinephrine must be used most judiciously or not at all."

In cardiac arrest a much bigger dose of Adrenalin in the heart is needed to make it start beating. The reaction of the anoxic heart to Adrenalin is delayed. Thus a big initial dose is needed. The action of Adrenalin is too short to cause any untoward effect once the heart is beating again.



A. *B.*

Fig. 1.—*A*, Case 3, a 57-year-old man. Roentgenogram taken on July 13, 1951, showed right-sided thick-walled tuberculous empyema. On July 21, 1951, a 5-rib thoracoplasty with apicolysis was performed; *B*, Case 3. This was the roentgenographic appearance when the circulatory arrest occurred. Twelve days later still a small left-sided pneumothorax remained after the three intracardiac injections.

Fig. 2*A*.—Case 4, a 51-year-old woman. Roentgenogram taken on Aug. 17, 1951, showing extensive left-sided tuberculosis, fixation of the left diaphragm, and a thickened left pleura.

Fig. 2*B*.—Case 4, roentgenogram taken on Dec. 29, 1951, three weeks after a first stage 3-rib thoracoplasty with apicolysis.

Fig. 2*C*.—Case 4, the roentgenographic appearance when the circulatory arrest occurred. The second stage thoracoplasty with removal of ribs 4 to 7 was just completed.

If the heart, however, is beating slowly, no pulse being palpable, the smaller dose of 1 ml. may be sufficient and should therefore always be given as the first injection. If no immediate response to that dose is noted, 5 ml. and, if necessary, 10 ml. Adrenalin, 1:1000, should then be injected with the needle left in place in the heart to save time.

This plan has then been followed in all cases except those in which, due to the nature of the surgical procedure, the direct cardiac massage is a quicker procedure.

CASE 4.—A 51-year-old woman with extensive left-sided tuberculosis had a first stage thoracoplasty with removal of the upper 3 ribs and apicolysis done Dec. 7, 1951. Four weeks later a second stage with removal of posterior parts of ribs 4, 5, 6, 7, and 8 was completed (see roentgenogram, Fig. 2). The second stage was done under epidural anesthesia with 50 ml. 1 per cent Xylocain. Sixty minutes after completion of the anesthesia, when the operation was finished, there was a fall in blood pressure. No blood was available for an immediate transfusion. In spite of intravenous nor-adrenalin the blood pressure continued to fall rapidly until no pulse could be felt and the patient stopped breathing. (1) She was immediately turned on her back, (2) 6 ml. Adrenalin, 1:1000, was injected intracardially through the left fourth intercostal space, (3) there was an immediate response with return of the heart action and a blood pressure of 160, (4) the patient was still bluish and without spontaneous breathing so artificial ventilation was instituted through an intratracheal tube, (5) after about 1 hour spontaneous breathing returned, and (6) after 1½ hours the patient had regained consciousness. The aftercourse was uneventful.

This 51-year-old woman had extensive tuberculosis in her left lung, diaphragm fixation, and thickened pleura after a left pleurisy. When the circulatory arrest occurred after the second stage thoracoplasty, the chest surgeon did not judge it to be possible for him to open this left chest and perform cardiac massage within 3 minutes. Experience from extrapleural resections for tuberculosis under thoracoplasties has shown these incisions to be more time consuming. A right-sided thoracotomy with cardiac massage would not have been tolerated from a functional point of view (see roentgenogram, Fig. 2*C*).

In two more cases the efficiency of a big dose of Adrenalin intracardially to overcome circulatory arrest has been found.

CASE 5.—A 39-year-old woman with a residual tuberculous cavity under a 5 rib thoracoplasty underwent a right upper lobectomy with resection of the apical subsegment of lower lobe. The endobronchial anesthesia technique had been used with nitrous oxide, oxygen, and curare. At the end of the operation, when the skin was being sutured, 1.5 mg. Prostigmin and 0.5 mg. atropine was given. Two minutes later a copious amount of clear yellow fluid was pouring out from the lungs. The paitent was turned over on her back and aspiration was carried out through the tube. The surgeon had not experienced such an idiosyncrasy to Prostigmin before and instead of removing the tube and sucking out the bronchial tree through a bronchoscope he continued an ineffective suction through a small plastic catheter.

Circulatory arrest due to anoxia occurred. There was, however, a delay of more than 3 minutes before 5 ml. Adrenalin, 1:1000, was injected intracardially with an immediate return of heart action. Three and one-half hours later spontaneous breathing returned. Six hours after the operation the patient died without having regained consciousness. Post-mortem examination showed pulmonary edema but no hypertrophy of the right ventricle. Death was due to the anoxic brain damage.

CASE 6.—A 23-year-old man had a 5-rib thoracoplasty performed on the right side. A big hematoma developed in the apicolysis space compressing the right lung. One day after the operation the wound was reopened and the hematoma evacuated in local anesthesia. At the end of the procedure, circulatory arrest occurred. The right lung was atelectatic. Adrenalin and a suitable needle was not at hand until more than 4 minutes had elapsed. Five milliliters Adrenalin, 1:1000, was given intracardially with an immediate return of heart action. The patient died 8 hours later without having regained consciousness. Post-mortem examination did not show the origin of the bleeding.

SUMMARY

In cases of circulatory arrest the generally accepted treatment with thoracotomy and cardiac massage cannot be adopted as the method of choice in all types of cases.

1. The best method is always prevention when possible.

2. In many bilateral tuberculous cases the pathologic changes of lungs and pleura will make it impossible for a chest surgeon to open the thorax and perform cardiac massage within 3 minutes.

3. Due to the diminished respiratory reserve many tuberculous patients do not tolerate an unexpected thoracotomy.

4. In two such cases the author administered large doses of Adrenalin intracardially with survival of the patients. None had lasting neurologic symptoms.

5. In circulatory arrest the author now immediately injects 1 ml. Adrenalin, 1:1000, through a wide bore needle into the heart. If no immediate return of heart action is found, 5 ml. Adrenalin, 1:1000, is injected through the needle left in place in the heart. This dose has so far been sufficient in most cases. If no pulse is palpable after one minute an additional 10 ml. of Adrenalin, 1:1000, is injected.

6. If this treatment is instituted later than 3 minutes after the circulatory arrest, the result is most often a return of heart action in a patient with cerebral damage.

7. The author believes that direct heart massage should only be performed in cases where, due to the nature of the operation, this is the faster procedure.

8. As time is the most important factor in circulatory arrest the author believes that the most rapid procedure should be chosen. It is thought that more patients are saved by immediate intracardiac injection of large doses of Adrenalin, compared to the number lost by cardiac massage one minute too late or due to ventricular fibrillation.

REFERENCES

1. Ament, R., Papper, E. M., and Rovenstine, E. A.: Cardiac Arrest During Anesthesia, Ann. Surg. **134**: 220, 1951.
2. Barber, R. F., and Madden, J. L.: Resuscitation of the Heart, Am. J. Surg. **64**: 151, 1944.
3. Beck, C. S., and Mautz, F. R.: The Control of the Heart Beat by the Surgeon, Ann. Surg. **106**: 525, 1937.
4. Beck, C. S.: Resuscitation for Cardiac Standstill and Ventricular Fibrillation Occurring During Operation, Am. J. Surg. **54**: 273, 1941.

5. Beck, C .S., and Rand, H. J.: Cardiac Arrest During Anesthesia and Surgery, J. A. M. A. **141**: 1230, 1949.
6. Becker, A. H.: Cardiorespiratory Failure, Am. J. Surg. **83**: 127, 1952.
7. Björk, V. O.: Brain Perfusions in Dogs With Artifically Oxygenated Blood, Acta Chir. scand. Supp. **96**: 137, 1948.
8. Bost, T. C.: Cardiac Arrest During Anesthesia and Surgical Operations, Am. J. Surg. **83**: 135, 1952.
9. Cooley, D. A.: Cardiac Resuscitation During Operations for Pulmonic Stenosis, Ann. Surg. **132**: 930, 1950.
10. Dripps, R. D., Kirby, C. K., Johnson, J., and Erb. W. H.: Cardiac Resuscitation, Ann. Surg. **127**: 592, 1948.
11. Ehrenhaft, J. L., Eastwood, D. W., and Morris, L. E.: Analysis of Twenty-seven Cases of Acute Cardiac Arrest, J. THORACIC SURG. **22**: 592, 1951.
12. Fauteux, M.: Cardiac Resuscitation, J. THORACIC SURG. **16**: 623, 1947.
13. Lahey, F., and Ruzicka, E. R.: Experiences With Cardiac Arrest, Surg. Gynec., and Obst. **90**: 108, 1950.
14. Nicholson, J. C.: Cardiac Massage, Brit. M. J. **1**: 385, 1942.
15. Ruzicka, E. R., and Nicholson, M. J.: Cardiac Arrest Under Anesthesia, J. A. M. A. **135**: 622, 1947.

ATELECTASIS: PHYSIOPATHOLOGY AND TREATMENT

Special Reference to Its Occurrence in Bulbar Poliomyelitis, and to a Suggested Therapeutic Aid by the Intrabronchial Administration of Trypsin

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ATELECTASIS has long been recognized as a pathologic process. Traube, in 1846, was able to collapse a lung or lobes of a lung by direct mechanical obstruction of a bronchus through creation of an artificial plug with shot or gum arabic.[22] The writings of Pasteur[18] describe numerous cases which resulted from paralysis of the diaphragm during diphtheritic infection. Other reports have appeared in the literature, but it is only within very recent years that practical application of this information has led to more enlightened methods of therapy. Jackson,[15] in 1925, was the first to correlate the clinical and experimental findings and successfully to cure an atelectasis by bronchoscopic aspiration of a diphtheritic membrane. Haight,[12] twelve years later, stressed the significance of atelectasis as an etiologic factor in postoperative bronchopneumonia. During the last war particular progress was made toward the general awareness of this problem through the experience of Burford, Burbank, Sampson[6] and others with the traumatic wet lung.

The recognition of atelectasis as a significant complication in poliomyelitis is also fairly recent. Sutherland,[21] in 1913, was the first to mention collapse of the lung among the complications of this disease. Regan,[19] eleven years later, again reported instances of this occurrence. Subsequently, twenty years elapsed before further reference was made to this pathologic process. At this time, Cooperstock[8] discussed the condition and recognized it as a definite complication of poliomyelitis. Perhaps the late cognizance of this complication is due to the fact that improved care over the last decade has considerably decreased the immediate mortality from bulbar poliomyelitis. Previously, the great majority of these patients did not live long enough to develop atelectasis. Since the care of these patients is still improving, we can anticipate that the problem of atelectasis in respiratory poliomyelitic cases will continue to increase.

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It would seem worth while, therefore, to discuss the etiology of atelectasis, and to emphasize particularly the problem as it presents in patients with bulbar poliomyelitis.

PATHOPHYSIOLOGY

Three major requisites are necessary for development of atelectasis in a lung, lobe, or segment. First, some mechanical cause for obstruction must be present. Second, there must be a sufficient circulation to allow absorption of the air distal to the obstruction. Third, there must be no accessory means for air entry into the distal portion of the obstructed area.

Normally, the body guards against the occurrence of atelectasis by several physiological mechanisms. Alteration in any one of these factors may contribute to its occurrence or may influence the severity of resulting signs and symptoms. These factors may be enumerated as follows: (1) efficient ciliary action on a normal bronchial mucous membrane; (2) a normal exudate from the bronchial mucosa; (3) an efficient collateral respiration; (4) a normally functioning process for disposal of secretions once they reach the pharynx; (5) an efficient cough mechanism; (6) absence of infection in the tracheobronchial tree.

Normally, secretions are removed by continuous ciliary activity, together with the expulsive action of bronchial inspiratory and expiratory movement. The ciliary action begins proximal to the respiratory bronchioles, but in the presence of allergy, chronic irritation, chronic infection and asthma, a metaplasia of the ciliated epithelium may occur,[14] thereby eliminating normal ciliary activity. Under such circumstances abnormal or excessive secretions are aided in their removal by the cough mechanism and by lymphatic absorption.

The effectiveness of ciliary action depends to a large extent on the viscosity of the secretions. The bronchial glands secrete both mucin and serous fluid. Buhrmester[5] has demonstrated that there is a direct relationship between the mucin content and the viscosity of secretions. In the presence of chronic irritation, inflammation, or allergy, there is an increased production of mucin with consequent increase in viscosity and adhesiveness of the secretions. Dehydration also will lead to an increased mucin content. Similarly, the type of infecting organism present in the secretions is a consideration in some instances. For example, pneumococcic sputum is extremely viscous.[9] It is also worth recognizing that sputum coughed up is not necessarily an index of the type of bronchial mucus or exudate occluding a segment or lung. It may rather represent an overflow from the mouth of the bronchus of the affected lobe, even though the bronchus itself is completely occluded. Further, the concept of obstruction of the large bronchi by discrete, essentially solid plugs is rarely found in actual practice. Instead, the occlusion of small bronchi and bronchioles by viscid secretions is more generally seen.

The third factor in this series of mechanisms is an efficient collateral respiration. Otherwise one would expect segmental or subsegmental atelectasis to occur more commonly. This is especially true in poliomyelitis, where

large and small bronchi are often filled with mucus which may be present some time before being suctioned off, if removed at all. Moreover, if this were not the situation, one would expect every bronchitis to lead to a spreading patchy atelectasis by the plugging of the smaller bronchi, thereby preventing the passage of air to the distal portions of the segment. Generally, air distal to the obstruction can assist in the expulsion of mucus plugs in the act of coughing, thus preventing eventual suffocation.[13]

The existence of collateral respiration was discovered by Van Allen in 1930 in association with Adams and Lindskog.[24, 26] It implies that the pulmonary lobules are not end organs from the standpoint of ventilation.[16] It has been demonstrated that after the direct bronchial route for air is obstructed artificially, there still exists an indirect and peripheral route for exchange and maintainance of aeration from adjacent ventilated segments. However, obstruction of a main bronchus would prevent air from entering an entire lobe, and therefore such collateral ventilation would not occur. Since collateral respiration is not dependent on collateral circulation, it tends to lessen the incidence of obstructive atelectasis, equalize pressure changes in the lung, equalize alveolar gas tension of O_2 and CO_2, and provide a more uniform oxygenation of venous blood. There is some evidence to show that the pores of Kohn, small anatomic defects in the alveolar walls, may provide the route for such exchange.

The cough mechanism is in itself dependent on many factors. First, the ability of the respiratory muscles to function; second, the presence of air in the lung tissue; third, the ability of the glottis to open and close; and fourth, the presence of a normal central cough center, a function that is not well understood.

Heinbecker,[23] in 1927, showed clearly that during respiration the bronchi change their caliber, being larger during inspiration and smaller during expiration. Recently Di Rienzo[10] has confirmed those observations. From these studies it has been conceived that a cough is a dynamic act of the mucous membrane that expels air or secretions by means of a high-speed peristaltic wave originating in the small bronchi and ending in the larynx. This is accompanied by the harmonic movement of the functional sphincters. This peristaltic bronchial wave may in itself move along secretions, but the greater the aeration distal to the secretions, the more effective will the cough be in removing any obstruction. Again, abnormal pulmonary conditions, such as infection or bronchiectasis, will cause excessive accumulation of secretions due to interference with these dynamic bronchial properties.

In poliomyelitis, the cough mechanism may be impaired either centrally or peripherally by paralysis of the muscles involved, such as the diaphragms, abdominal, and intercostal muscles. Also, paralysis of the larynx will prevent the normal abduction and adduction of the cords necessary for the build-up and release of intrathoracic and intrapulmonary pressure required in the act of coughing. Poor bronchial drainage from the hypoventilated pulmonary tissue present in respiratory poliomyelitis cases will lead to a reduction in size

and mobility of the bronchi. Also, a reduction in vital capacity will vitiate against an effective cough.[8]

A defective or absent cough in the poliomyelitic patient is usually accompanied by difficulty in disposing of secretions once they have reached the pharynx or throat, and these cases require prompt suction for the removal of these secretions. Similarly, in those patients with tracheostomies, any delay in aspiration from the trachea may lead to further stagnation of the secretions with consequent increase in viscosity, or to subsequent reinhalation of the secretions into distal bronchi or bronchioles.

The obstructing agent in poliomyelitis is either from bronchial secretions or from aspirated exogenous material. In either case, this material is infected and the severity of the atelectasis will to a great extent be dependent upon the degree of infection present. Coryllos[9] placed a sterile obstructing balloon into the bronchi of dogs and left it there for sixteen to twenty-four hours. During this period atelectasis ensued, with slight fever, increased respiration, cough, and typical radiographic findings. After removal of the balloon, gradual reaeration in from sixteen to twenty-four hours took place. When the bronchus was obstructed with an infected balloon or with plugs of sputum containing bacteria of varying virulence, it was noted that the severity of symptoms as well as the mortality varied with the virulence of the organisms. Moreover, the infection seemed to spread from the site of obstruction through the bronchial lymphatics and into the distal lung parenchyma, setting up a consolidated segment which could lead to abscess formation or even gangrene. If infection set in, the possibility of reaeration became less, even though the primary obstructing factor was removed. It is accepted, however, that a variable amount of alveolar exudate is absorbed from the lungs over a period of time. Head[13] states that when the parenchyma distal to an obstructed segment or lobule becomes opaque on x-ray film, it does so because of consolidation and not primarily because of airlessness. In poliomyelitis, atelectasis often follows infections of the upper respiratory tract, and the implication of this as far as the application of therapy is obvious. Again the atelectasis may be slow or rapid, with or without obvious signs and symptoms (see Case 2).

The length of time a segment of lung may remain collapsed, and yet still be reaerated, is a debatable point, but in general depends on the presence and degree of infection. Under vigorous antibiotic therapy, infection to all intents and purposes may be eliminated, and with the removal of the obstructing agent, complete reabsorption of the parenchymatous exudate may take place within a short time. As early as 1903 Ascoli and associates[1] and later Bittorf[4] observed that liquefaction of parenchymatous exudate was greatly aided by the liberation of two proteolytic enzymes from the white blood cells in the exudate. If infection occurs in the distal segment, irreversible pathologic changes may appear within a short while. If infection has not occurred, and it is often difficult to determine this, re-expansion may be obtained after many months, if not longer.

THERAPY

The understanding of the above factors in the etiology and pathogenesis of atelectasis allows formulation of a reasonable approach to therapy. Generally, the fault does not lie with a single factor, but more often is due to a combination of several or all of them. It is the rare case where one can remove a single obstruction or plug, and following this have the lung return to normal. Such an example is the removal of a foreign body soon after its aspiration. In respiratory poliomyelitis cases there may be a combination of diminished respiratory function, absent or defective cough, inability to dispose of pharyngeal secretions, abnormal bronchial secretions in quantity and quality, plus respiratory infection. One sometimes finds that these patients may also have had asthma or chronic bronchitis, which has led to a damaged bronchial mucous membrane. In such cases there is generally need for more than simple aspiration of the larger bronchi, although rarely spontaneous recovery without any specific therapy may occur. Cooperstock[8] reports that three of his four cases cleared on sulfanilamide therapy alone. We have had one instance in which re-expansion occurred five months after development of a right upper-lobe atelectasis.

Many methods have been described for the prophylaxis of this condition (deep breathing exercises, inhalation of high concentration of CO_2, and intravenous iodide therapy), but these methods are useful in short-term therapy such as postoperative states. In general, they are impracticable in the chronic poliomyelitic. In the respiratory poliomyelitic, intermittent positive pressure therapy and the oscillating bed have been advocated, but proof of their effectiveness is not convincing.

The management of the patient who has developed atelectasis should be directed at satisfying the following conditions: (1) removal of the obstructing agent; (2) loosening and removal of viscid bronchial secretions; (3) establishment of an efficient collateral respiration; (4) aid in the removal of the viscid tracheal and pharyngeal secretions; (5) assistance in the securing of a good cough; (6) treatment of any incipient infection.

Bronchoscopic aspiration, as indicated above, does not necessarily remove all the secretions in the smaller bronchi and bronchioles, but, by opening up the larger proximal and segmental branches, allows collateral respiration to be re-established. Once suction of these bronchi has been performed, insufflation of the affected lung with oxygen under slight pressure through the bronchoscope may aid in getting some air into the distal obstructed areas.

Atelectasis of long standing may be aided considerably by loosening of the viscid secretions. Baker and associates[2] advocate the use of iodides, which they have demonstrated experimentally to increase the amount of serous exudate by direct stimulation of the bronchial glands or by the alteration of the cellular permeability of the bronchial mucosa. They injected 1 to 2 Gm. of sodium iodide intravenously and noted by means of bronchoscopy a great increase of bronchial secretions occurring in one to fifteen minutes and lasting two to four minutes, depending on the amount of iodide injected. They treated 11 cases with postoperative atelectasis who had received pre- and postoperative

antiobiotics with 1 to 2 Gm. of sodium iodide intravenously twice a day for 3 to 4 days after onset of the atelectasis. These patients were ambulated early and encouraged to cough. By this means the condition cleared within twenty-four to forty-eight hours. Generally, in the poliomyelitic patient this therapy is not advisable, as it only will increase the amount of secretion with which the patient is already having difficulty. The same would apply to aerosol therapy with antispasmodics, which Christie and co-workers[7] have found so effective in their cases. They report that the aerosol inhalation of 1 per cent epinephrine and 1 per cent ephedrine (in a proportion of 1:9), or Neo-Synephrine five to eight times a day, releases bronchospasm. The patient, if then given an expectorant mixture to loosen up secretions, can often rid himself of all secretions and relieve the atelectasis.

Recently there has been considerable interest in the use of various enzymatic agents for physiologic débridement. There are currently available three such products: Tryptar,* Varidase,† and Caroid.‡ Muenster and associates[17] have recently reported on the successful intrabronchial instillation of streptokinase and streptodornase in a long-standing case of atelectasis. There was no clearing of a left lower lobe atelectasis following three bronchoscopies, so one month later they instilled 20,000 units of streptokinase plus 9,500 units of streptodornase in 5 c.c. of solution into the left lower lobe bronchus. The patient responded by coughing up thin watery secretions. The procedure was repeated the following day with double the above dosages, and, on bronchoscopy, it was noted that there was no mucosal change. There was a slight elevation of temperature, and on the third day there was clinical and radiological improvement following the coughing up of these secretions.

The product Tryptar has attracted our attention for two reasons, (1) it is an enzyme occurring normally in the human body, and (2) there is within normally living human cells an antitrypsinoginase which effectively prevents its action except on dead or injured tissues. Investigation has also demonstrated that mucoid material from man is effectively dissolved by the action of trypsin. Such physiologic activity seemed to us to justify the application of purified crystalline trypsin to the management of certain difficult cases of chronic atelectasis unrelieved by the usual methods of therapy. Ideal candidates for such a study are those patients with severe bulbar poliomyelitis who secondarily develop persistent atelectasis.

Roettig and associates[20] reported on the use of trypsin in chest diseases and noted that other than a histamine-like reaction which occurred with the intrapleural injection of this substance in cases of empyema, and which could be prevented by adequate antibiotic therapy, the procedure was relatively safe. They also found that in tuberculosis empyemata, the number of tubercle bacilli rapidly decreased. It has been postulated that this may be

*Armour Laboratory's preparation of purified crystalline trypsin. Grateful acknowledgment is made to this organization for supplying this material as used in the clinical and experimental observations.

†Lederle Laboratory's preparation of streptokinase-streptodornase.

‡American Ferment Company's preparation of the vegetable enzymes papaya and papayatin obtained by fermentation from Carica Papaya.

due to the action of trypsin on the tubercle capsule, thereby making the organism susceptible to ordinary antibiotics.[11] Roettig treated 10 cases by aerosol trypsin, giving 100 mg. in 2 c.c. of Sorensen's Phosphate Buffer solution twice a day. He noted a decrease in the viscosity of secretions and sputum, but these cases did develop a slight irritation of the tracheobronchial tree as well as hoarseness. We have given trypsin endobronchially to four patients with atelectasis, two of whom had poliomyelitis. These poliomyelitic patients were carefully selected because of the severity of their basic pathology, and because their prognosis seemed so poor without the aid of something more definitive being accomplished, bronchoscopy alone having failed to remove the obstruction. In most instances therapy was carried out while the patient was in the Monaghan respirator. Initially, small doses of Tryptar were utilized but, as confidence in the procedure increased, a standard dilution was established. This consisted of 250 mg. of trypsin in 5 c.c. of Sorensen's Phosphate Buffer solution.

The procedure used was to place the patient in a semireclining position at about 20 degrees and propped at about 30 degrees with the affected side down. The bronchoscope was then introduced and all endobronchial secretions aspirated from both main stem bronchi and all orifices. Subsequently, the bronchoscope was passed into the main stem bronchus of the dependent side; trypsin solution was slowly introduced through the branch oxygen inlet of the bronchoscope. By slow introduction, the trypsin was aspirated into all segmental bronchial orifices. The scope was then removed and the patient allowed to remain in the same position for one hour. The bronchoscope was once again introduced and all secretions completely aspirated.

Bronchoscopy in our poliomyelitic patients has not required anesthesia because they all have had tracheostomies.

One of the major problems in the respiratory poliomyelitic invalid is the inability to cough efficiently in order to bring up any secretions. Barach and associates[3] describe the use of an exsufflator which can be adapted to the tank-type respirator, or to a pressure hood, from which a high volume flow rate of air during expiration can be achieved, and which would be equivalent to a moderately vigorous cough. This is done by the gradual withdrawal of air until a negative pressure of about 40 mm. Hg is obtained, and then suddenly releasing the pressure so that it rises to atmospheric pressure within a 0.04 second. The rate is set from six to eight times a minute to prevent hyperventilation with the pressures used, and to prevent impairment of cardiac output. This high degree of inflation of the lung produced by 40 mm. Hg negative pressure has the added effect of increasing the diameter of bronchi and may facilitate the passage of air beyond the obstructing mucin or mucus plugs, thereby increasing the effectiveness of the expiratory blast in pushing the secretions into the larger bronchi, trachea, and pharynx. It is suggested that three to five courses of treatment of one-half hour a day for two to five days may be required to accomplish complete aeration of the lungs and disappearance of atelectasis. The procedure is stopped at ten-minute intervals to allow removal of secretions by suction.

Perhaps the use of this exsufflator following endobronchial instillation of trypsin would aid greatly in the handling of cases of long-standing atelectasis.

Finally, one must control infection. It has been our policy to treat any upper or lower respiratory tract infection in our poliomyelitic cases vigorously with antibiotics, and in the eventuality of a suspected or proved atelectasis to give large doses of penicillin. If throat or laryngeal cultures demonstrate organisms that may be more sensitive to other antibiotics, these may also be used.

The suctioning and aid to the patient in the removal of secretions from the upper respiratory tract is part and parcel of normal nursing, but, following attempts to relieve an atelectasis as described above, particularly after the instillation of trypsin or other drugs which loosen or stimulate secretions, special attention and repeated suctioning for some time will be necessary.

Following the bronchoscopic aspiration and other therapy advocated, we feel that the encouragement of deep breathing either by means of strong respiratory pressures, or, better still, by means of a positive pressure apparatus for ten to thirty minutes three to five times a day is useful, particularly if one precedes this by prior antispasmodic aerosol therapy. In our wards we have found Isyprel, five drops in 20 drops saline given as an aerosol through the oxygen tubing, particularly useful. The exsufflator, if available, could be used daily, and should be a great help in the prevention of respiratory complications in poliomyelitis.

The following two case reports illustrate the use of trypsin instilled endobronchially in cases of chronic atelectasis.

CASE REPORTS

CASE 1.—B. S., a 31-year-old white woman, was admitted to Colorado General Hospital on Nov. 5, 1950, with acute bulbar spinal polimoyelitis. Following admission she was placed in a respirator, and during the next five-month period her condition improved sufficiently so that she could be gradually weaned from auxiliary respiratory aid. Examination on discharge from the hospital revealed a severe paralysis of all four extremities, but most marked in the arms. There was some weakness of the abdominal and back muscles, and there was slight weakness of the left diaphragm; the right diaphragm was paralyzed, and there was severe weakness of all intercostal muscles. She weighed at this time about 75 pounds. Past history revealed that she had suffered from bronchial asthma for many years, apparently aggravated by her social environment. The patient did not have any attacks of asthma while in the hospital, but described such episodes on the few occasions when she was home on week-end leave. On Jan. 10, 1952, the patient had a rather severe asthmatic attack. She was hospitalized in her home community and treated with oxygen and antiasthmatic drugs without any benefit. Subsequently, she was again transferred to Colorado General Hospital where examination revealed an extremely apprehensive and anxious patient in spite of previous large doses of sedatives. Her respirations were rapid and shallow, and she complained of dyspnea with difficulty in breathing. There was very little movement of the chest cage, and all respiration seemed mainly left abdominal. Breath sounds were diminished over the entire chest, and were absent over the right lower chest. Very few rhonchi or râles were audible. It was felt that the patient was not having an asthmatic attack, but had developed a right lower-lobe atelectasis. Fluoroscopy was performed and revealed atelectasis of the middle lobe and portions of the right lower lobe (Fig. 1). The patient continued to develop increasing dyspnea and seemed unable to dispose of the accumulating secretions. Consequently, two

days following admission, a tracheostomy was performed. Almost immediately her color improved, but her breathing remained labored. She was placed in an Emerson respirator and for a few hours each day on the oscillating bed. This treatment was supplemented with antibiotics and intermittent positive pressure oxygen. She also received aerosol Isyprel and postural drainage. However, her condition did not improve on this conservative management. She continued to run a low-grade fever and to have difficulty carrying on respiration without aid. On January 16, bronchoscopy was performed (Fig. 2). All segmental orifices were observed and noted to be normal. There was some accumulated secretion in the left lower-lobe orifice which was aspirated. The bronchoscope was removed to the carina where, under positive pressure oxygen, the lungs were inflated. Subsequently, there was no change in the clinical or roentgenological findings of the patient. On January 25, a repeat bronchoscopy was performed (Fig. 3). The right main bronchus, below the level of the upper-lobe bronchus, was found to be partially occluded by a large quantity of thick, white, mucoid secretion. This material was aspirated as fully as possible so that the middle- and lower-lobe orifices would be well visualized. Again there was no improvement clinically or radiographically. Bronchoscopy was again repeated on January 30. A large, thick plug of

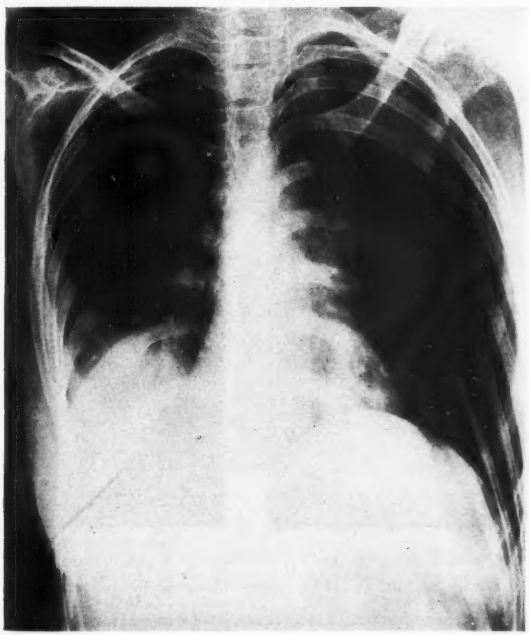

Fig. 1.—Roentgenogram, Jan. 10, 1952, reveals atelectasis of right middle lobe and portions of right lower lobe.

yellowish secretion in the right main bronchus was observed to occlude the right middle-lobe and right lower-lobe bronchi. A very thorough aspiration was performed, but again clinical and roentgenological findings remained unchanged. On February 4, it was decided to re-bronchoscope the patient and to instill 125 mg. of Tryptar in 5 c.c. of Sorensen's Phosphate Buffer solution. Prior to installation of this solution into the right main stem bronchus a small quantity of tenacious creamy secretion was aspirated. The bronchial tree was found to be markedly erythematous as well as somewhat edematous around the upper-lobe orifice.

One hour later bronchoscopy revealed small amounts of much less viscid secretion in the right main and lower-lobe bronchi. This was aspirated so that all orifices were observed unobstructed by any secretions. The patient tolerated the procedure well, and subsequently seemed somewhat improved clinically. Fluoroscopy revealed apparent clearing of the right middle lobe without change in the appearance of the right lower lobe. The same procedure was carried out again on Feb. 7. On this occasion a small amount of thick mucoid secretion was observed in the right upper-lobe orifice and right main stem bronchus just below the middle-lobe orifice. None of the lobar bronchi appeared to be occluded, however. One hour following the introduction of 125 mg. of Tryptar a small amount of frothy mucoid secretion was seen in the right lower-lobe bronchus. This was aspirated well, after which a good blast of air was obtained from beyond this area. Clinically, the

Fig. 2.

Fig. 3.

Fig. 2.—Roentgenogram, Jan. 16, 1952, taken following bronchoscopy, reveals a persistent atelectasis of the right lower lobe.

Fig. 3.—Roentgenogram, Jan. 25, 1952, taken following second bronchoscopy, reveals no improvement in atelectatic process.

patient showed no change in physical signs over the previous procedure. On February 11 the patient was bronchoscoped once more and again a large amount of thick creamy yellowish secretion was found in the right bronchus. This was again aspirated, and on this occasion 250 mg. of Tryptar in 5 c.c. of Sorensen's Phosphate Buffer solution were injected slowly into the right bronchus. One hour later a considerable quantity of thin watery mucoid secretion was removed. This appeared to be coming from the right lower lobe. The opinion was expressed that the patient's bronchial tree had shown considerable improvement during the recent courses of therapy with Tryptar. Roentgenographic examination of the

chest on February 15 (Fig. 4) showed no evidence whatsoever of atelectasis, and clinically the patient was much improved. Fluoroscopy on February 22 revealed a paralyzed right diaphragm with good movement of the left diaphragm. The costophrenic sinus on the right was somewhat obscured, but the lung was clear. It was felt that she had some chronic pleuritic thickening. There was paradoxical movement of the right diaphragm on sneezing and coughing. This was more marked in the erect than in the supine position. The patient was bronchoscoped again on March 7, at which time the right bronchus appeared normal. Roentgenographic examination at this time again showed no evidence of atelectasis (Fig. 5). The patient was then discharged.

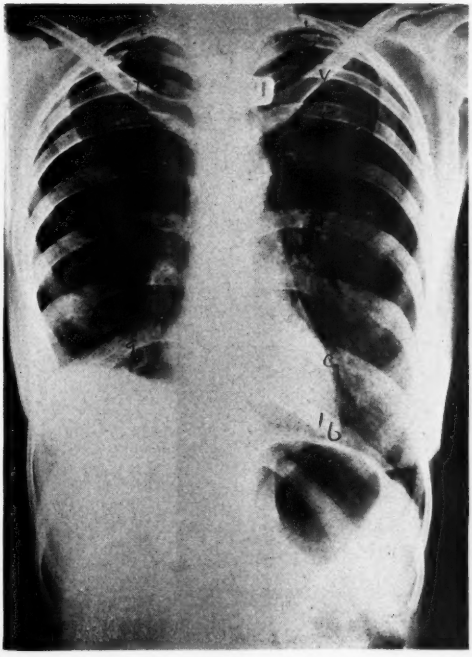

Fig. 4.

Fig. 5.

Fig. 4.—Roentgenogram, Feb. 15, 1952, taken following endobronchial trypsin therapy, reveals disappearance of atelectasis.

Fig. 5.—Lateral chest roentgenogram from March 9, 1952, series shows no evidence of residual pathology or any latent effects from endobronchial trypsin.

CASE 2.—C. O., a 34-year-old white man, was admitted to Colorado General Hospital on Aug. 19, 1951, with the diagnosis of poliomyelitis. He complained of respiratory difficulty and general weakness of all extremities. His illness began three days previously with nausea, vomiting, vertigo, and some headache. Examination revealed in particular that there was equal expansion of the chest and that both diaphragms were functioning. However, the intercostals appeared to be paralyzed. The patient had increasing difficulty with respiration because of inability to dispose of excessive tracheal secretions. Consequently, on the day following admission tracheostomy was performed. During the next two days the disease progressed, to result in complete paralysis of all extremities, the back, and the abdominal muscles. The patient also developed difficulty in swallowing. On August 24 he had an acute episode of respiratory distress. He was cold, clammy, appre-

hensive, and cyanotic. He showed definite mental symptoms with facial grimacing and profuse salivation. These were attributed to cerebral hypoxia. Since it was impossible to remove the patient from the respirator even momentarily, the clinical signs of atelectasis were difficult to evaluate. It was felt, however, that this was the basic cause of the patient's difficulty. Deep tracheal suction was apparently inadequate. Therefore, bronchoscopy was performed and both main stem bronchi were cleared of a large amout of mucopurulent sputum with resultant improvement in the patient's aeration and color. Three days later this procedure was repeated and again revealed a large amount of mucous material particularly on the left side. Removal of these mucus plugs resulted again in marked clinical improvement.

Fig. 6.—Roentgenogram taken Nov. 26, 1951, following the fourth bronchoscopy, reveals persistent atelectasis of the left lower lobe.

The patient showed little evidence of recovery from his paralyses during the next two-month period. He was able to be out of the Emerson respirator for no longer than fifteen minutes. Examination on these occasions revealed dullness over both lower lung fields. On Nov. 16, 1951, the patient developed sudden difficulty in breathing and a rapid, thready pulse. Roentgenographic examination revealed haziness of the entire left lung field with some mediastinal shift to the left. Bronchoscopic aspiration was again performed with subsequent improvement in the patient's general clinical condition. Three days later the patient was fluoroscoped and was observed to have hyperventilation of the right lung. There was some scoliosis with a concavity to the left. Auscultation revealed dullness over the left lower-lung field. It was felt that the atelectasis contributed in large extent to the inability of the patient to remain out of a respirator. Repeated bronchoscopy on November 21 allowed aspiration of two small mucus plugs from the left lower bronchus, but the patient showed no clinical improvement (Fig. 6). A chest roentgenogram, taken December 27, revealed atelectasis of the left lower lobe still present, with a marked shift of the mediastinum and heart to the left. The right lung now showed definite signs

of compensatory emphysema. The patient showed some slight improvement during the next month. He was able to be out of the respirator for twenty-eight to thirty minutes. Examination, however, still did not reveal any significant movement of the diaphragm or chest. A check roentgenogram of the chest was obtained on Jan. 25, 1952, and now showed an essentially complete atelectasis of the entire left lung (Fig. 7). Examination revealed dullness and absent breath sounds corresponding to this area. It was apparent that there had been a clinically silent extension of the atelectatic process during the time since the roentgenogram one month previously. The patient had not complained of any increased dyspnea, nor did he show any elevation of temperature or other signs associated with an atelectatic process. On January 30 the patient was bronchoscoped once more. Although the original atelectasis had been present for at least five months and the upper lobe collapse present for an indeterminate period of time, we felt that it would be worth trying

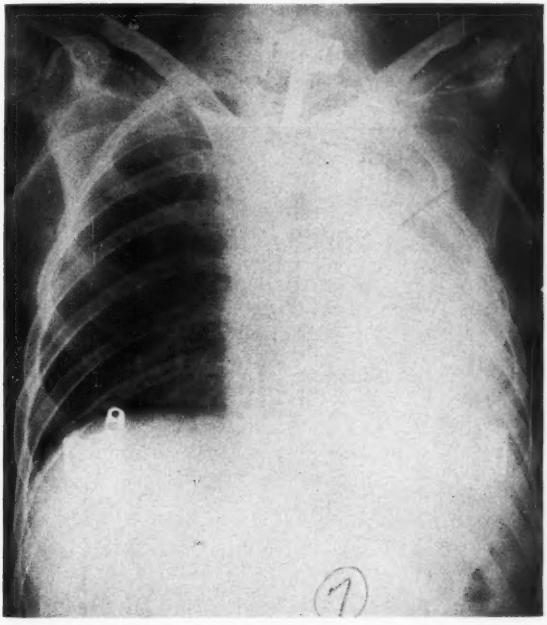

Fig. 7.—Roentgenogram taken Jan. 25, 1952, reveals extension of atelectasis process with essentially complete atelectasis of left lung.

the instillation of Tryptar in an attempt to loosen any plugs which might be occluding major bronchi. At bronchoscopy a considerable amount of mucous material was aspirated from the tracheobronchial tree. It was noted that the patient breathed only with the right lung, although he was in a Monaghan respirator at this time. Following aspiration, 125 mg. of Tryptar in 5 c.c. of Sorensen's Phosphate Buffer solution were introduced slowly into the left bronchus. The patient was allowed to remain in a semireclining position on his left side for one hour. He was then rebronchoscoped and all secretions removed from the tracheobronchial tree. Subsequently, it was observed that the patient was now breathing with both sides of the chest. His color showed considerable improvement, as noted by the nurse on the ward who had been taking care of him. Clinically, there was again evidence of air entry into the left upper lung field. Approximately four hours following Tryptar instillation the patient developed chills and fever up to 103° F. His breathing was not

dyspneic, but he did complain of slight pain in the left chest and some burning in the trachea. He was immediately given 50 mg. of Benadryl intravenously, and within two hours all symptoms had disappeared. Roentgenologic examination of the chest now showed that the entire upper lobe had cleared but that the atelectasis of the lower lobe remained. Subsequently, roentgenograms on January 31 (Fig. 8), February 2, and February 4 did not reveal any further clearing of this area of atelectasis at the left base. However, the patient was able to increase his period out of the respirator to as much as forty-five minutes, and his general condition showed marked improvement. One month later a roentgenogram revealed condensation of the atelectatic area of the left base, so that now there existed only a small triangular shadow behind the heart (Fig. 9). The impression was that there had been a marked improvement over the previous roentgenograms. On March 10 it was decided to bronchoscope the patient again and on this occasion to inject 250 mg. of Tryptar. Following this procedure, a considerable amount of thin, watery mucous secretion was removed from the left lower-lobe bronchus (Fig. 10). During the following week the patient was able to increase the period of time out of a respirator to as much as one and one-half hours, and in two weeks to as much as three hours. Roentgenograms have shown essentially complete resolution of the remaining atelectatic area (Fig. 11).

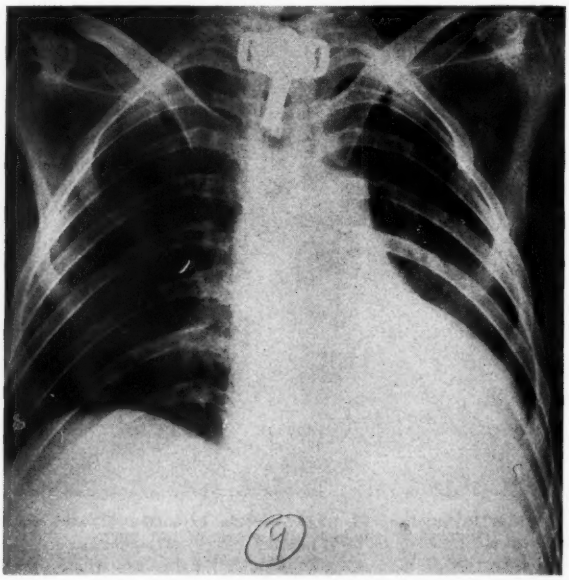

Fig. 8.—Roentgenogram taken Jan. 31, 1952, following initial trypsin therapy, illustrates clearing of left upper-lobe atelectasis with no improvement in right lower-lobe collapse.

DISCUSSION

It would seem logical to assume that chronic atelectasis should be attacked more vigorously from a conservative standpoint before the stage is reached which will require radical surgical measures. The use of the physiologic enzyme, trypsin, appears to offer this possibility. Trypsin has been shown to have a mucolytic action in vitro and might reasonably be expected to aid in the removal of inspissated bronchial secretions which have resulted

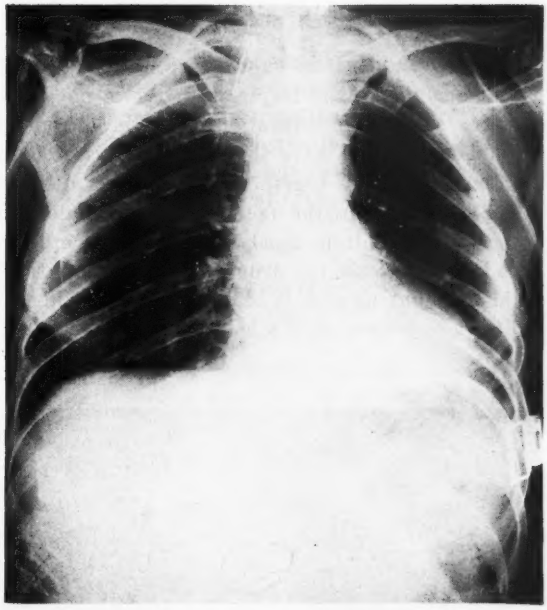

Fig. 9.—Roentgenogram taken March 6, 1952, illustrates the improvement, but partial atelectasis of the left lower lobe persists.

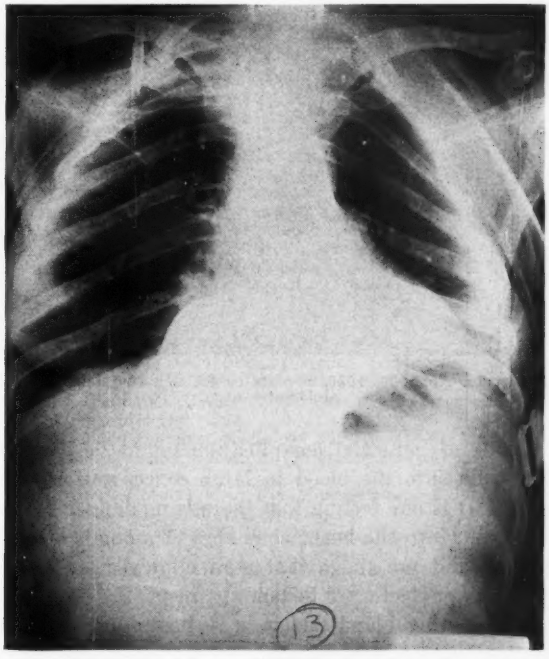

Fig. 10.—Roentgenogram, March 12, 1952, following second endobronchial administration of trypsin, illustrates the progressive improvement in the atelectatic process.

in atelectasis. Since it would not be expected to have any effect on the bronchial mucosa, local damage to the respiratory tree is not to be anticipated. However, in the face of acute inflammation of the bronchial tree, some local tissue damage could occur. We have shown that in tissue culture the fibrin matrix holding young fibroblasts together is dissolved and the cells are killed by trypsin in large dosage. In the face of an acute inflammatory process trypsin could, therefore, result in local edema and superficial necrosis. It would perhaps be best not to use the drug in cases of acute tracheobronchitis. Experience has also taught us that it is contraindicated in tuberculosis if a bronchopleural fistula is present or if a fistula is opened as a result of trypsin administration.

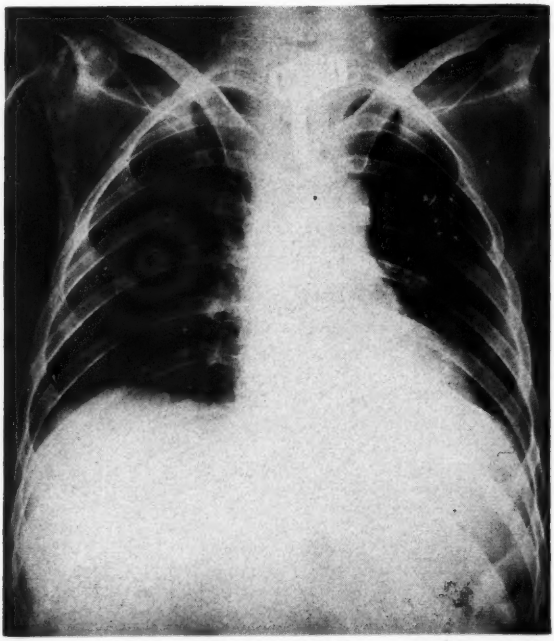

Fig. 11.—Roentgenogram, April 29, 1952, reveals essentially complete resolution of remaining atelectatic area.

Another source of potential complication might be related to the rapid absorption of trypsin into the blood in large concentrations from the pulmonary parenchyma. It is our feeling that there is no danger in this regard. We have injected trypsin into the heart chambers of animals (rats) in concentrations equivalent to and far above that used clinically, without any evidence of damage or of anaphylactic or histaminic response. Antitrypsinogenase is present in the circulating blood and it is to be assumed that the activity of trypsin is destroyed almost immediately.

We assumed, therefore, and have shown, that the procedure of endobronchial administration of trypsin is an innocuous one physiologically, and that it is worthy of more thorough investigation in carefully selected cases of chronic atelectasis due to bronchial obstruction from inspissated mucoid material. We feel that the problem of atelectasis in bulbar poliomyelitis offers the ideal situation in which this type of therapy might be beneficial.

SUMMARY

1. The physiopathology of atelectasis is reviewed.

2. The therapy of atelectasis is considered, particularly in the patient with respiratory poliomyelitis.

3. The use of trypsin by endobronchial installation is described as it has been used in loosening the viscid secretions of patients with atelectasis.

4. Two case reports are presented illustrating the successful use of this material in relieving chronic atelectasis due to bronchial and bronchiolar obstruction in patients with bulbar poliomyelitis.

5. It is urged that further experience be accumulated on the effectiveness of endobronchial trypsin in relieving persistent atelectasis in carefully selected patients.

Acknowledgment is made to Dr. James J. Waring for his advice and counsel, and to Dr. Gertrude Weiss, Dr. Edward B. Liddle, and Mr. Lloyd H. Hurley for their assistance in various aspects of the clinical and experimental studies referred to in this paper.

REFERENCES

1. Ascoli, M., and Bezzola, C.: Das verhalten dos Antitryptischen Vermogens des Blutserums bei der Grouposen Pneumonie, Berl. Klin. Wchnschr. **40**: 391, 1903.
2. Baker, J. N., Roettig, L. C., and Cuntes, G. M.: The Prevention and Treatment of Atelectasis by the Control of Bronchial Secretions, Ann. Surg. **134**: 641, 1951.
3. Barach, A. L., Beck, G. J., Bickerman, H. A., and Seanor, H. E.: Mechanical Coughing: Studies on Physical Methods of Producing High Velocity Flow Rates During the Expiratory Cycle, Trans. A. Am. Physicians **64**: 360, 1951.
4. Bittorf, A.: Ueber die Verteilung des Proteolytischen Leukozytenferments und seines Antiferments in Harn, Blut und Auswurf im Verlaufe der Krupposen Pneumonie, Deutches Arch. f. Klin. Med. **91**: 212, 1907.
5. Buhrmester, C. C.: Nasal Mucin, Ann. Otol. Rhin. and Laryng. **45**: 687, 1936.
6. Burford, Thomas H., and Burbank, Benjamin: Traumatic Wet Lung: Observations on Certain Physiologic Fundamentals of Thoracic Trauma, J. Thoracic Surg. **14**: 415, 1945.
7. Christie, H. E., Meakins, J. F., and Aronovitch, M.: Aerosol Therapy in the Treatment of Postoperative Pulmonary Atelectasis, Canad. M. A. J. **61**: 388, 1949.
8. Cooperstock, M.: Atelectasis Complicating Acute Poliomyelitis with Involvement of Respiratory Muscles, Am. J. Dis. Child. **67**: 457, 1944.
9. Coryllos, P. N., and Birnbaum, G. L.: Syndrome of Pneumococcal Bronchial Obstruction, Arch. Int. Med. **51**: 290, 1933.
10. Di Rienzo, S.: Bronchial Dynamism, Radiology **53**: 168, 1949.
11. Dye, William: Personal communication (Fitzsimons Army Hospital, Denver, Colorado).
12. Haight, C.: Intratracheal Suction in the Management of Postoperative Pulmonary Complications, Ann. Surg. **107**: 218, 1938.
13. Head, J.: In Postscript to Shaw R. R.: Mucoid Impaction of Bronchi, J. Throacic Surg. **22**: 149, 1951.
14. Hilding, A. C.: The Relation of Ciliary Insufficiency to Death from Asthma and Other Respiratory Diseases, Ann. Otol. Rhin. and Laryng. **52**: 5, 1943.
15. Jackson, C.: Bronchoscopy and Esophagoscopy and Gastroscopy, Ed. 2, Philadelphia, W. B. Saunders Co., p. 301.
16. Lindskog, G. E.: Collateral Respiration in Normal and Diseased Lung, Yale J. Biol. and Med. **23**: 311, 1951.

17. Muenster, J. J., Flance, I. J., and Sweeny, B.: Treatment of Unresolved Pneumonia With Streptokinase and Streptodornase, Am. J. Med. **12**: 376, 1952.
18. Pasteur, W.: Respiratory Paralysis After Diphtheria as a Cause of Pulmonary Complications with Suggestions as to Treatment, Am. J. Med. Sc. **100**: 242, 1890.
19. Regan, J. C.: Massive Collapse of the Lung in Acute Poliomyelitis, Lancet **2**: 1222, 1924.
20. Roettig, L. C., Reisser, H. G., Habeeb, W., and Mark, L.: The Use of Trypsin in Chest Disease, Dis. Chest. **21**: 245, 1952.
21. Sutherland, G. A.: Case of Anterior Poliomyelitis; Paralysis of Abdominal Muscles, Collapse of Lung, Proc. Roy. Soc. Med. Land. **7**: 31, 1913-14.
22. Traube, L.: Ges. Beitr. z. Path. u. Physiol. **1**: 65, 1846.
23. Heinbecker, P.: A Method for the Demonstration of Calibre Changes in the Bronchi in Normal Respiration, J. Clin. Investigation **4**: 459, 1927.
24. Van Allen, C. M.: Collateral Respiration in the Lung: Râle in Bronchial Obstruction to Prevent Atelectasis and to Restore Patency, Surg., Gynec. & Obst. **53**: 16, 1931.
25. Van Allen, C. M., and Adams, W. E.: Mechanism of Obstructive Atelectasis, Surg., Gynec. & Obst. **50**: 385, 1930.
26. Van Allen, C. M., and Lindskog, G. E.: Obstructive Atelectasis; Problems of Pathogenesis and Clinical Management, Arch. Surg. **21**: 1195, 1930.

CONCURRENT LYMPHOMA OF THE LUNG AND STOMACH

Follow-up on a Previously Reported Case

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THE case described herein has previously been reported in this journal as primary lymphoma of the lung.[1] Since that publication what was originally thought to be an associated benign gastric ulcer in an otherwise normal stomach has proved to be a recurrent ulcer forming in a stomach that had diffuse lymphomatous change. It would appear, therefore, that this patient had two lymphomas at the same time. This has been definitely established by operation both in the stomach and the lung, and both seem to be following a benign course. This case is not one of single primary lymphoma of the lung. It may be a double primary tumor such as is seen at times in an epithelial neoplasm or one may be metastatic from the other. It is for this reason and the unusual general interest of this case that we make the following report. This case also represents to our knowledge the first published account of a lymphoma of the stomach diagnosed by gastroscopic biopsy and proved by operation.

PAST HISTORY

A 47-year-old housewife was first admitted to the Guthrie Clinic on Nov. 8, 1949, complaining of dull, aching epigastric pain of two months' duration. She had similar episodes at intervals for the past fifteen years, but in the past two months the distress was more severe and almost constant. Most foods aggravated her distress, while antacids gave her some relief. There was occasional nausea, never vomiting. She had lost 10 pounds in the previous two months. Three weeks before admission she became "lightheaded" and had short-lived "peculiar abdominal pain." The following day, and for several days thereafter, she passed black-colored feces. There were no pulmonary symptoms. Physical examination showed a moderately obese woman with mild epigastric tenderness. There were no other significant abnormalities noted. Extensive laboratory studies showed only a mild anemia. A caffeine fractional gastric analysis was within normal limits. A routine chest roentgenogram showed a discrete, smoothly rounded opacity in the lower lobe of the right lung near the costophrenic angle about 6 cm. in diameter (Fig. 1). Gastroduodenal x-ray series revealed a defect along the greater curvature posterior surface which was pliable and changed with peristaltic movements. No crater was seen by fluoroscopy, though it was demonstrated on the roentgenogram (Fig. 2). The examination was repeated one week later, at which time the same findings were apparent, though less marked. A gastroscopy was performed and showed an ulcer in the lower third of the stomach on the posterolateral aspect. The edges were ragged and irregular, but not rolled or raised. A near-by patch of atrophic gastritis was well visualized. The gastroscopic biopsy of the ulcer showed the degenerated cellular exudate

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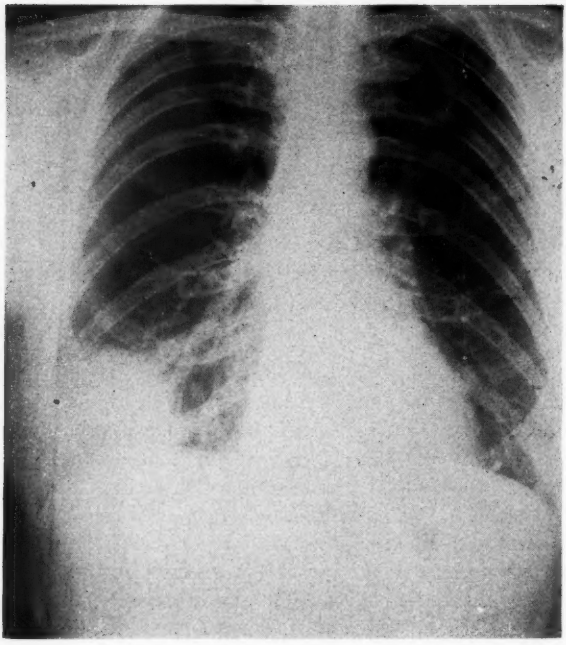

Fig. 1.—Chest roentgenogram showing right lower-lobe tumor.

Fig. 2.—Gastroduodenal roentgenogram showing ulcer defect of posterior wall.

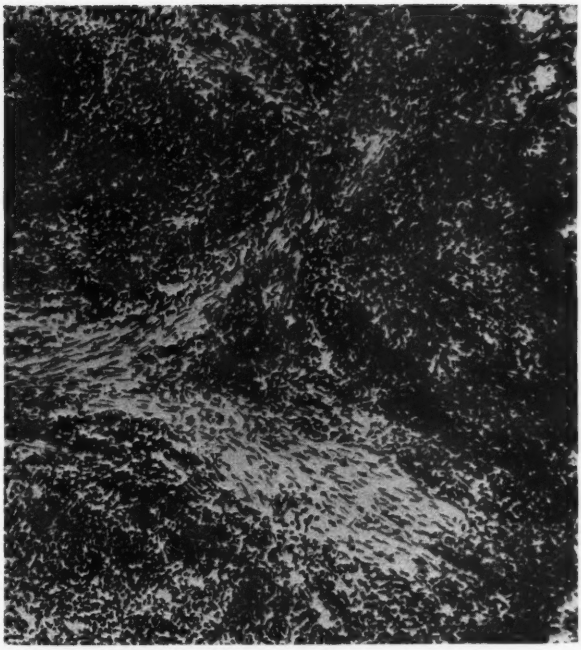

Fig. 3.—Biopsy of ulcer showing a few typical gastric glands and massive cellular infiltration, first interpreted as probably inflammatory; later thought to be a fairly typical lymphoma.

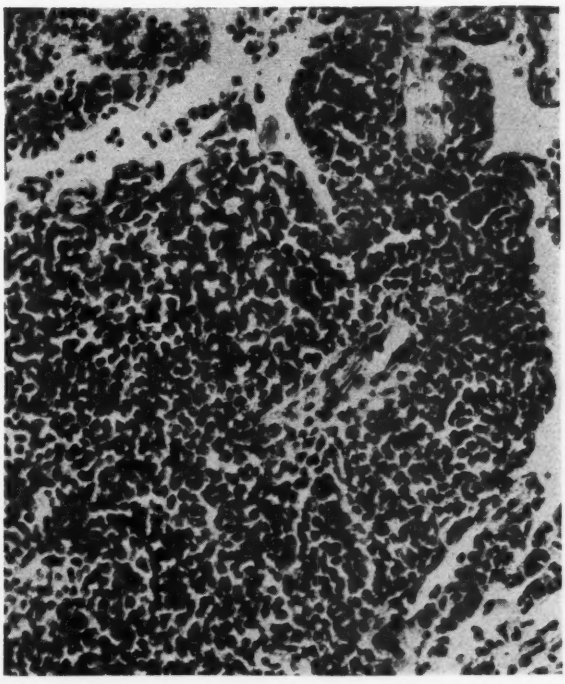

Fig. 4.—Microscopic section of lung tumor showing a typical lymphoma.

typical of that found in the base of any ulcer (Fig. 3). The cells showed no mitotic activity, and it was thought that this tissue represented benign gastric mucosa with considerable cellular infiltration and reaction, and a degenerating ulcer membrane.

It was believed by the surgeon that the lung tumor was probably metastatic from a malignant process in the stomach. It was decided that the lung be explored first. A thoracotomy revealed a firm mass palpable in the lower lobe of the right lung. There were no adhesions present, but the tumor was firmly encapsulated. There were no other tumors found. A right lower lobectomy was performed. Microscopic examination of the

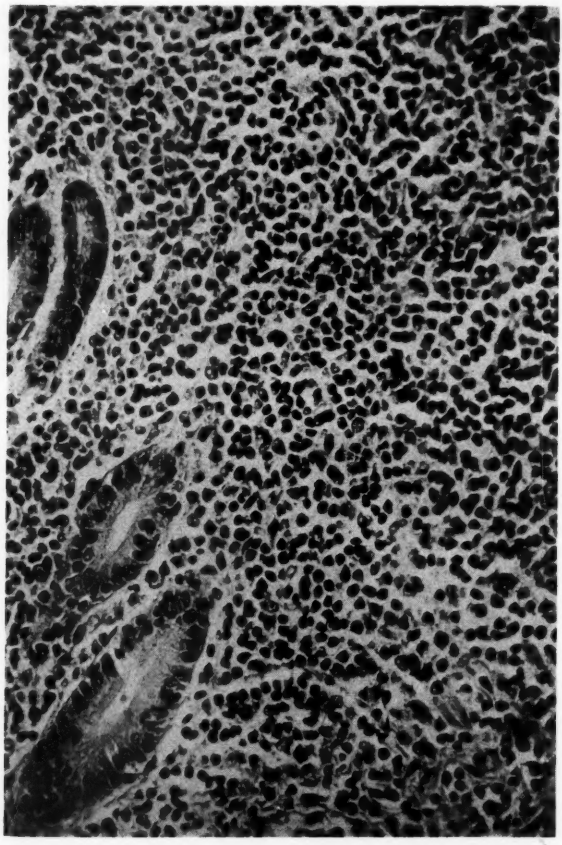

Fig. 5.—Microscopic section of stomach after subtotal gastrectomy, showing change identical to that of the lung tumor—lymphoma.

tumor showed that it consisted almost completely of round cells without stroma (Fig. 4). It was believed that this was probably a benign lymphoma, and the ulcer of the stomach was benign and not connected with the lung lesion or related to it.

The patient made an uneventful recovery. Her gastrointestinal symptoms subsided. During her convalescence a gastroscopic examination was done and another stomach roentgenogram made. Both showed remarkable and complete healing of the gastric ulcer. Repeated roentgenograms throughout 1950 and 1951 showed no recurrence of the ulcer, and the chest remained clear.

PRESENT ADMISSION

The patient was again seen here in December, 1951, having had some epigastric distress at irregular intervals for several weeks, without vomiting, but with the passage of black stools for several days. Examination showed a pale, haggered-appearing patient with mild epigastric tenderness. Otherwise the findings were essentially normal. Her hemoglobin was 10 Gm. per hundred cubic centimeters, and dropped to 7.5 in the next two days. Stomach roentgenogram again showed a thickened irregularity of the greater curvature, not truly rigid, and without apparent crater. At gastroscopy, an ulcer about the size of a 25 cent piece was seen; it was covered with mucus, and had clean-cut edges. A Papanicolaou stain of these secretions was negative for tumor cells, and a gastroscopic biopsy showed round-cell infiltration and marked inflammatory reaction. A subtotal gastric resection was performed. Gallstones were found at exploration. The surgeon believed it was unwise to remove them at this time. The stomach was markedly thickened throughout, and there were many hemorrhagic areas. No frank ulcerations were seen. A frozen section microscopic examination showed a stomach section to have been replaced almost entirely by round cells, having the exact appearance of the lymphoma removed from the lung three years previously (Fig. 5). Permanent section examination confirmed this finding. The patient had an uneventful postoperative recovery, and has had no distress up to this time.

DISCUSSION

Many questions arise that cannot be answered definitely: Is this a malignant or benign condition? The tumor does not appear malignant either by gross or microscopic examination, and the patient's clinical course so far is benign. Marshall and Brown[2] have reported many patients who have survived for years with this disease. They find that the prognosis is generally better with lymphomas of the stomach than with carcinomas of the stomach. Is this a primary lymphoma of the stomach or lung? Did they develop simultaneously? Are they merely part of a generalized lymphomatous condition? One thing seems definite. Lymphoma of the stomach was diagnosable by gastroscopic biopsy three years ago. Review of the gastroscopic biopsy specimens removed at that time showed a round-cell infiltration which was mistakenly called gastritis. In light of three years of additional experience with gastroscopic biopsy, we believe that this tissue could definitely be diagnosed as lymphoma.

SUMMARY AND CONCLUSIONS

A case of lymphoma of the stomach and lung has been presented.

This case was previously reported as one of primary lymphoma of the lung. It is now apparent that it was not a single primary tumor.

In retrospect, we believe that a correct diagnosis could have been made three years previously by proper interpretation of the gastroscopic biopsy.

REFERENCES

1. Beck, W. C., and Reganis, J. D.: Primary Lymphoma of the Lung, J. THORACIC SURG. **22**: 323-328, 1951.
2. Marshall, S. F., and Brown, L.: Primary Malignant Lymphoid Tumors of the Stomach, Surg. Clin. North America **30**: 885-892, 1950.

Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 33rd annual meeting of the American Association for Thoracic Surgery will be held March 27, 28, and 30, 1953, in San Francisco, Calif. Headquarters will be at the Hotel Fairmont. Suitable accommodations also have been reserved at near-by hotels, the Mark Hopkins, the Huntington and the Sir Francis Drake.

Requests for Hotel Reservations

These should be mailed to Dr. H. Brodie Stephens, Chairman of Hotel Committee, American Association for Thoracic Surgery, Room 200, 61 Grove St., San Francisco 2, Calif. The following information must be included: (1) two choices of hotels; (2) type of accommodation desired—single, double, twin, room or suite; (3) name of other room occupants, if any, whether wife or another doctor; (4) date and approximate hour of arrival; (5) date of departure; (6) return address.

Abstracts for Papers

Abstracts for the presentation of papers at the 1953 meeting must be received on or before Dec. 1, 1952, otherwise they will not be considered by the Program Committee. *Five, repeat, five* copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words, and should accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the American Association for Thoracic Surgery must be received by the Membership Committee not later than December 1, 1952, to be considered for action at the meeting in San Francisco in March, 1953. Applications received on a date beyond that deadline must be deferred for consideration until 1954.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors must submit a separate letter concerning the applicant direct to the Chairman of the Membership Committee. These regulations are at the direction of the Council and were made effective at the meeting in Dallas in 1952.

Completed application forms should be sent to the Chairman of the Membership Committee, Edward M. Kent, M.D., 1406 Clark Building, Pittsburgh 22, Pa.

INDEX TO VOLUME 24

A

Abbott, Osler. Discussion carcinoma of esophagus, 268
Discussion detecting changes of blood supply to heart, 115
Discussion experimental pulmonary venous occlusion, 199
Discussion resection of trachea for carcinoma, 244
Discussion rupture of esophagus, 383
Discussion surgical management of pulmonary emphysema, 408
Discussion traumatic saccular aneurysm of thoracic aorta, 344
Ada, Alexander. Discussion of hemopericardium and constrictive pericarditis, 367
Adenoma, adenocarcinoma developing in peripheral bronchial (Umiker and Storey), 420
Adenoma, obstructive pneumonitis secondary to bronchial (McBurney, Clagett, and McDonald), 411
Alexander, John. Discussion of nodular tuberculosis, 575
Alley, Ralph D. Discussion of experimental pulmonary venous occlusion, 199
American Association for Thoracic Surgery, notice of 33rd annual meeting, 212, 324, 434, 544, 642
Anderson, Robert L. Rupture of esophagus, 369, 388
Aneurysm of thoracic aorta, traumatic saccular (Hollingsworth, Johnston, and McCooey), 325
Anterior mediastinal tracheotomy (Kleitsch), 38
Artificial heart problem, some physiologic aspects of (Dodrill, Hill, and Gerish), 134
oxygenation and circulation during complete by-pass of heart (Helmsworth, Clark, Kaplan, Sherman, and Largen), 117
Atelectasis on pulmonary blood flow in dog, effects of (Peters and Roos), 389
physiopathology and treatment (Peck and Levin), 619
Atresia of cervical esophagus (Johnson and Mangiardi), 75
Aves, Fred H. (with Desforges, Gerard, and Strieder, John W.) Mercury-bag bolus to expedite passage of esophageal dilator guide string, 34

B

Bacteria on tracheobronchial trees of young rats, effects of increased endobronchial pressure, Vitamin A deficient diets, and nasopharyngeal and installations of (O'Neill, Bradshaw, Hightower, and Ward), 80
Bahnson, Henry T. (with Ravitch, Mark M., and Johns, Thomas N. P.) Carcinoma of esophagus, 256
Beck, William C. (with Shallenberger, Paul L., Fisher, Peter, and DeWan, Charles H.) Concurrent lymphoma of lung and stomach—follow-up on previously reported case, 637
Becker, Barney B. (with Murphy, James D., and Swindell, H. V.) Complications and results of treatment of bronchopleural fistula following resection for tuberculosis, 578, 586
Berman, Jacob K. (with Test, Pasquale S., and McArt, Bruce A.) Congenital esophagobronchial fistula in an adult, 493
Berry, Frank B. Waste of slaughter and rage of fight—presidential address, 1
Björk, Viking Olov. Circulatory arrest, 611
Blood flow in dog, effects of atelectasis on pulmonary (Peters and Roos), 389
supply to heart, experimental methods for detection of changes of (Goldman, Shaw, Corday, Fields, Kennamer, Smith, Lindgren, and Prinzmetal), 105
Bolton, Houck E. (with Maniglia, Rosario, and Massey, Franklin C.) Calcific emboli complicating mitral valve commissurotomy, 502
Bradshaw, H. H. (with O'Neill, James F., Hightower, Felda, and Ward, D. E., Jr. Effects of increased endobronchial pressure, vitamin A deficient diets, and nasopharyngeal installations of bacteria on tracheobronchial trees of young rats, 80
Brantigan, Otto C. Discussion of plastic sponge prosthesis after resection, 607
Discussion of resection of trachea for carcinoma, 245
Discussion of technique of producing mitral stenosis of controlled degree, 164
Brewer, Lyman A., III. Discussion of carcinoma of esophagus, 266
Bronchial adenoma, adenocarcinoma developing in peripheral (Umiker and Storey), 420
obstructive pneumonitis secondary to (McBurney, Clagett, and McDonald), 411
Bronchiectasis in infants, some aspects of (Mendez, Leahy, and Butsch), 50
Bronchiogenic carcinoma, a product of multiple sites of origin (McGrath, Gall, and Kessler), 271
silicotic and tuberculosilicotic lesions simulating (Kergin), 545

Bronchitis, acute tuberculous fibrinous (Williams), 530
Bronchography, indications for delayed roentgenogram in (Kane and Heiser), 70
Bronchopleural fistula following resection for tuberculosis, complications and results of treatment of (Murphy, Becker, and Swindell), 578
Bronchus, fibrosarcoma of—report of three cases diagnosed by bronchoscopy and treated by resection (Storey), 16
Burdette, Walter J. Induced pulmonary tumors, 427
Burke, Donald R. (with Hanlon, C. Rollins, and Sabiston, David C., Jr.) Experimental pulmonary venous occlusion, 190
Butsch, Winfield L. (with Mendez, Fernando L., Jr., and Leahy, Leon J.) Some aspects of bronchiectasis in infants, 50

C

Calcific emboli complicating mitral valve commissurotomy (Bolton, Maniglia, and Massey), 502
Carcinoma, a product of multiple sites of origin, bronchiogenic (McGrath, Gall, and Kessler), 271
of esophagus (Ravitch, Bahnson, and Johns), 256
complicating cardiospasm (Gore and Lam), 43
of hypopharynx, larynx, and upper esophagus, surgical treatment of (Grimes and Stephens), 246
resection of trachea (Cotton and Penido), 231
trans-sternal tracheal excision for (Minor), 88
Carcinosarcoma, endobronchial (Taylor and Rae), 93
Cardiac wounds, healing of extensive (Thomas and Ziffren), 346
Cardiospasm, carcinoma of esophagus complicating (Gore and Lam), 43
Carswell, James, Jr. Discussion of treatment of bronchopleural fistula, 585
(with Shields, D. O., Chapman, John S., Jr., and Wollenman, O. J.) Nodular tuberculosis, 568
Carter, Max G. Discussion of surgical management of pulmonary emphysema, 409
Chapman, John S., Jr. (with Shields, D. O., Carswell, James, Jr., and Wollenman, O. J.) Nodular tuberculosis, 568, 577
Chest management of war wounds of (Valle), 457
Circulatory arrest (Björk), 611
Clagett, O. Theron. Discussion of plastic sponge prosthesis after resection, 609
Discussion of resection of trachea for carcinoma, 242
Clagett, O. Theron—Cont'd
(with McBurney, Robert P., and McDonald, John R.) Obstructive pneumonitis secondary to bronchial adenoma, 411
(with McDonald, John R., and Schmidt, Herbert W.) Localized fibrous mesothelioma of pleura, 213
(with Swift, Edward A., and Grindlay, John H.) Repair of tracheal defects with fascia and tantalum mesh: an experimental study, 482
Clark, Leland C., Jr. (with Helmsworth, James A., Kaplan, Samuel, Sherman, Roger T., and Largen, Thomas) Artificial oxygenation and circulation during complete by-pass of heart, 117
Coburn, Donald (with Fisher, Edwin R., and Effler, Donald B.) Thymic neoplasms, 58
Condon, William B. Discussion of localized fibrous mesothelioma of pleura, 229
Congenital esophagobronchial fistula in an adult (Berman, Test, and McArt), 493
Cooley, Denton A. Discussion of traumatic saccular aneurysm of thoracic aorta, 343
Corday, Eliot (with Goldman, Alfred, Shaw, Clinton, Fields, Joshua, Kennamer, S. Rexford, Smith, Allen, Lindgren, Inga, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105
Cotton, Bert H. (with Penido, Joao R. F.) Resection of trachea for carcinoma, 231, 245
Crenshaw, Gerald L. (with Rowles, Donald F.) Surgical management of pulmonary emphysema, 398, 410
Curreri, Anthony R. (with Gale, Joseph W., Young, William P., and Dickie, Helen A.) Plastic sponge prosthesis following resection in pulmonary tuberculosis, 587

D

Daniel, Rollin A. Discussion of traumatic saccular aneurysm of thoracic aorta, 345
Daniels, C. F. Discussion of use of Sk-Sd (Varidase) in management of early postoperative partial pulmonary resections, 302
Davidson, Louis R. Discussion of treatment of bronchopleural fistula, 584
Davis, Edgar. Discussion of plastic sponge prosthesis after resection, 607
De Bakey, Michael. Discussion of use of Sk-Sd (Varidase) in management of early postoperative partial pulmonary resections, 303
Desforges, Gerard (with Aves, Fred H., and Strieder, John W.) Mercury-bag bolus to expedite passage of esophageal dilator guide string, 34

Deterling, Ralph A., Jr. Discussion of traumatic saccular aneurysm of thoracic aorta, 344
DeWan, Charles H. (with Shallenberger, Paul L., Fisher, Peter, and Beck, William C.) Concurrent lymphoma of lung and stomach—follow-up on previously reported case, 637
Dickie, Helen A. (with Gale, Joseph W., Curreri, Anthony R., and Young, William P.) Plastic sponge prosthesis following resection in pulmonary tuberculosis, 587
Dodrill, F. D. (with Hill, Edward, and Gerisch, Robert) Some physiologic aspects of artificial heart problem, 134, 152
Drainage bottles, rack for underwater (Steele), 323

E

Effler, Donald B. (with Fisher, Edwin R., and Coburn, Donald) Thymic neoplasms, 58
Ehrenhaft, Johann L. (with Taber, Rodman E.) Hemopericardium and constrictive pericarditis, 355, 368
Ellison, Robert G. (with Major, Robert C., Pickering, M. S., and Hamilton, Wm. F.) Technique of producing mitral stenosis of controlled degree, 154, 164
Emboli, complicating mitral valve commissurotomy, calcific (Bolton, Maniglia, and Massey), 502
Emphysema, localized pulmonary hypertrophic (Williams), 522
  surgical management of pulmonary (Crenshaw and Rowles), 398
Endobronchial carcinosarcoma (Taylor and Rae), 93
  pressure, vitamin A deficient diets and nasopharyngeal installations of bacteria on the tracheobronchial trees of young rats, effects of increased (O'Neill, Bradshaw, Hightower, and Ward), 80
Esophageal dilator guide string, mercury-bag bolus to expedite passage of (Aves, Desforges, and Strieder), 34
  lesions, further discussion of right thoracic approach to (Hollingsworth), 201
Esophagobronchial fistula in an adult, congenital (Berman, Test, and McArt), 493
Esophagus, atresia of cervical (Johnson and Mangiardi), 75
  carcinoma of (Ravitch, Bahnson, and Johns), 256
  complicating cardiospasm, carcinoma of (Gore and Lam), 43
  rupture of (Anderson), 369
  surgical treatment of carcinoma of hypopharynx, larynx, and upper (Grimes and Stephens), 246

F

Fascia and tantalum mesh, repair of tracheal defects with (Swift, Grindlay, and Clagett), 482
Ferrara, Joseph F. (with Litwak, Robert S., Gadboys, Howard L., and Scott, George B.) Surgical approach for stenotic lesions of semilunar valves by excision and cusp replacement under direct vision, 165
Fibrosarcoma of bronchus—report of three cases diagnosed by bronchoscopy and treated by resection (Storey), 16
Fields, Joshua (with Goldman, Alfred, Shaw, Clinton, Corday, Eliot, Kennamer, S. Rexford, Smith, Allen, Lindgren, Inga, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105
Finnerty, J. J. Discussion of atelectasis and pulmonary blood flow in dog, 395
Fisher, Edwin R. (with Coburn, Donald, and Effler, Donald B.) Thymic neoplasms, 58
Fisher, Peter (with Shallenberger, Paul L., Beck, William C., and DeWan, Charles H.) Concurrent lymphoma of lung and stomach—follow-up on previously reported case, 637
Fistula in an adult, congenital esophagobronchial (Berman, Test, and McArt), 493
Fox, Robert T. Discussion of plastic sponge prosthesis after resection, 608
Fryfogle, J. D. Discussion of rupture of esophagus, 387

G

Gadboys, Howard L. (with Litwak, Robert S., Scott, George B., and Ferrara, Joseph F.) Surgical approach for stenotic lesions of semilunar valves by excision and cusp replacement under direct vision, 165
Gale, Joseph W. (with Curreri, Anthony R., Young, William P., and Dickie, Helen A.) Plastic sponge prosthesis following resection in pulmonary tuberculosis, 587, 610
Gall, Edward A. (with McGrath, Edward J., and Kessler, Dale P.) Bronchiogenic carcinoma, a product of multiple sites of origin, 271
Gerisch, Robert (Dodrill, F. D., and Hill, Edward) Some physiologic aspects of artificial heart problem, 134
Gibbon, John H., Jr. Discussion of carcinoma of esophagus, 269
  Discussion of symposium on artificial heart problem, 151

Goldman, Alfred. Discussion of silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma, 566
(with Shaw, Clinton, Corday, Eliot, Fields, Joshua, Kennamer, S. Rexford, Smith, Allen, Lindgren, Inga, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105, 116
Goodman, Walter N. (with Haas, Albert, and Rusk, Howard A.) Rehabilitation in thoracic surgery, 304
Gordon, Joseph. Discussion of nodular tuberculosis, 576
Gore, Ira (with Lam, Conrad R.) Carcinoma of esophagus complicating cardiospasm, 43
Gray, Fred. Discussion of treatment of bronchopleural fistula, 585
Grimes, Orville F. (with Stephens, H. Brodie) Surgical treatment of carcinoma of hypopharynx, larynx, and upper esophagus, 246
(with Thorburn, Jack D., and Stephens, H. Brodie) Benign thymoma in hilus of lung, 540
Grindlay, John H. (with Swift, Edward A., and Clagett, O. Theron) Repair of tracheal defects with fascia and trantalum mesh: an experimental study, 482

H

Haas, Albert (with Rusk, Howard A., and Goodman, Walter N.) Rehabilitation in thoracic surgery, 304
Hamilton, Wm. F. (with Ellison, Robert G., Major, Robert C., and Pickering, Raymond W.) Technique of producing mitral stenosis of controlled degree, 154
Hanlon, C. Rollins. Discussion of atelectasis and pulmonary blood flow in dog, 396
(with Sabiston, David C., Jr., and Burke, Donald R.) Experimental pulmonary venous occlusion, 190, 200
Harper, Frederick R. Discussion of symposium on war wounds of chest, 476
Heart, artificial oxygenation and circulation during complete by-pass of (Helmsworth, Clark, Kaplan, Sherman, and Largen), 117
experimental methods for detection of changes of blood supply to (Goldman, Shaw, Corday, Fields, Kennamer, Smith, Lindgren, and Prinzmetal), 105
problem, some physiological aspects of artificial (Dodrill, Hill, and Gerisch), 134
Heiser, Saul (with Kane, Irving J.) Indications for delayed roentgenogram in bronchography, 70
Helmsworth, James A. (with Clark, Leland C., Jr., Kaplan, Samuel, Sherman, Roger T., and Largen, Thomas) Artificial oxygenation and circulation during complete by-pass of heart, 117, 152
Hemopericardium and constrictive pericarditis (Ehrenhaft and Taber), 355
Heroy, William W. Discussion of rupture of esophagus, 384
Higginson, John F. Discussion of silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma, 566
Hightower, Felda (with O'Neill, James F., Bradshaw, H. H., and Ward, D. E., Jr.) Effects of increased endobronchial pressure, vitamin A deficient diets, and nasopharyngeal installations of bacteria on tracheobronchial trees of young rats, 80
Hill, Edward (with Dodrill, F. D., and Gerisch, Robert) Some physiologic aspects of artificial heart problem, 134
Hollingsworth, R. K. Further discussion of right thoracic approach to esophageal lesions, 201
(with Johnston, William W., and McCooey, James F.) Traumatic saccular aneurysm of thoracic aorta, 325
Hudson, Theodore R. Wound of superior vena cava with survival, 101
Hughes, Felix A., Jr. Discussion of experimental pulmonary venous occlusion, 199
Discussion of noduar tuberculosis, 576
Hypopharynx, larynx, and upper esophagus, surgical treatment of carcinoma of (Grimes and Stephens), 246

I

Infants, some aspects of bronchiectasis in (Mendez, Leahy, and Butsch), 50

J

Johns, Thomas N. P. (with Ravitch, Mark M., and Bahnson, Henry T.) Carcinoma of esophagus, 256
Johnson, E. K. (with Mangiardi, J. L.) Atresia of cervical esophagus, 75
Johnston, William W. (with Hollingsworth, R. K., and McCooey, James F.) Traumatic saccular aneurysm of thoracic aorta, 325

K

Kane, Irving J. (with Heiser, Saul) Indications for delayed roentgenogram in bronchography, 70

Kaplan, Samuel (with Helsworth, James A., Clark, Leland, C., Jr., Sherman, Roger T., and Largen, Thomas) Artificial oxygenation and circulation during complete by-pass of heart, 117

Kay, Earle B. Discussion of resection of trachea for carcinoma, 243

Kelley, Winfield O. (with Pecora, David V.) Resection in pulmonary tuberculosis, 510

Kennamer, S. Rexford (with Goldman, Alfred, Shaw, Clinton, Corday, Eliot, Fields, Joshua, Smith, Allen, Lindgren, Inga, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105

Kergin, Frederick G. Silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma, 545, 567

Kessler, Dale P. (with McGrath, Edward J., and Gall, Edward A.) Bronchiogenic carcinoma, a product of multiple sites of origin, 271

Kleitsch, William P. Anterior mediastinal tracheotomy, 38

Korea, care of thoracic and thoracoabdominal wounds in combat zone (Rockey), 435

L

Lam, Conrad R. (with Gore, Ira) Carcinoma of esophagus complicating cardiospasm, 43

Lambert, Adrian, Jr. Discussion of hemopericardium and constrictive pericarditis, 367

Largen, Thomas (with Helsworth, James A., Clark, Leland C., Jr., Kaplan, Samuel, and Sherman, Roger T.) Artificial oxygenation and circulation during complete by-pass of heart, 117

Larynx, and upper esophagus, surgical treatment of carcinoma of hypopharynx (Grimes and Stephens), 246

Leahy, Leon J., (with Mendez, Fernando L., Jr., and Butsch, Winfield L.) Some aspects of bronchiectasis in infants, 50

Leeds, Sanford E. Discussion of symposium on artificial heart problem, 151

Lees, William M. Discussion of treatment of bronchopleural fistula, 584

Levin, Stanley (with Peck, Mordant E.) Atelectasis: physiopathology and treatment, 619

Lindgren, Inga (with Goldman, Alfred, Shaw, Clinton, Corday, Eliot, Fields, Joshua, Kennamer, S. Rexford, Smith Allen, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105

Litwak, Robert S. (with Gadboys, Howard L., Scott, George B., and Ferrara, Joseph F.) Surgical approach for stenotic lesions of semilunar valves by excision and cusp replacement under direct vision, 165

Lundberg, Sven. Discussion of plastic sponge prosthesis after resection, 609

Lung and stomach, concurrent lymphoma of (Shallenberger, Fisher, Beck, and DeWan), 637

benign thymoma in hilus of (Thorburn, Stephens, and Grimes), 540

Lymphoma of lung and stomach, concurrent (Shallenberger, Fisher, Beck, and DeWan), 637

M

MacManus, J. E. Discussion of carcinoma of esophagus, 265

Discussion of resection of trachea for carcinoma, 244

Maier, H. C. Discussion of localized fibrous mesothelioma of pleura, 228

Major, Robert C. (with Ellison, Robert G., Pickering, Raymond W., and Hamilton, Wm. F.) Technique of producing mitral stenosis of controlled degree, 154

Mangiardi, J. L. (with Johnson, E. K.) Atresia of cervical esophagus, 75

Maniglia, Rosario (with Bolton, Houck E., and Massey, Franklin C.) Calcific emboli complicating mitral valve commissurotomy, 502

Mason, James M. III. Discussion of rupture of esophagus, 385

Discussion of silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma, 566

Massey, Franklin C. (with Bolton, Houck E., and Maniglia, Rosario) Calcific emboli complicating mitral valve commissurotomy, 502

McArt, Bruce A. (with Berman, Jacob K., and Test, Pasquale, S.) Congenital esophagobronchial fistula in an adult, 493

McBurney, Robert P. (with Clagett, O. Theron, and McDonald, John R.) Obstructive pneumonitis secondary to bronchial adenoma, 411

McCooey, James F. (with Hollingsworth, R. K., and Johnston, William W.) Traumatic saccular aneurysm of thoracic aorta, 325, 345

McDonald, John R. Discussion of bronchiogenic carcinoma, a product of multiple sites of origin, 283

(with Clagett, O. Theron and Schmidt, Herbert W.) Localized fibrous mesothelioma of pleura, 213

(with McBurney, Robert P., and Clagett, O. Theron) Obstructive pneumonitis secondary to bronchial adenoma, 411

McGrath, Edward J. (with Gall, Edward A., and Kessler, Dale P.) Bronchiogenic carcinoma, a product of multiple sites of origin, 271, 283
Mendez, Fernando L., Jr. (with Leahy, Leon J., and Butsch, Winfield L.) Some aspects of bronchiectasis in infants, 50
Mercury-bag bolus to expedite passage of esophageal dilator guide string (Aves, Desforges, and Strieder), 34
Mesothelioma of pleura, localized fibrous (Clagett, McDonald, and Schmidt), 213
Meyer, Herbert Willy. Discussion of rupture of esophagus, 385
Minor, George R. Trans-sternal tracheal excision for carcinoma, 88
Miscall, Laurence. Discussion of symposium on war wounds of chest, 477
Mitral stenosis of controlled degree, technique of producing (Ellison, Major, Pickering, and Hamilton), 154
Mulvihill, Daniel A. Discussion of treatment of bronchopleural fistula, 582
Murphy, James D. (with Becker, Barney B., and Swindell, H. V.) Complications and results of treatment of bronchopleural fistula following resection for tuberculosis, 578

N

Neoplasms, thymic (Fisher, Coburn, and Effler), 58

O

O'Neill, James F. (with Bradshaw, H. H., Hightower, Felda, and Ward, D. E., Jr.) Effects of increased endobronchial pressure, vitamin A deficient diets, and nasopharyngeal installations of bacteria on tracheobronchial trees of young rats, 80
Overholt, Richard H. Discussion of rupture of esophagus, 385

P

Peck, Mordant E. (with Levin, Stanley) Atelectasis: physiopathology and treatment, 619
Pecora, David V. (with Kelley, Winfield O.) Resection in pulmonary tuberculosis, 510
Penido, Joao R. F. (with Cotton, Bert H.) Resection of trachea for carcinoma, 231
Pericarditis, hemopericardium and constrictive (Ehrenhaft and Taber), 355
Peters, Richard M. (with Roos, Albert) Effects of atelectasis on pulmonary blood flow in dog, 389, 396
Pickering, Raymond W. (with Ellison, Robert G., Major, Robert C., and Hamilton, Wm. F.) Technique of producing mitral stenosis of controlled degree, 154
Pleura, localized fibrous mesothelioma of (Clagett, McDonald, and Schmidt), 213
Pneumonitis secondary to bronchial adenoma, obstructive (McBurney, Clagett, and McDonald), 411
Presidential address, Frank B. Berry, 1
Price Thomas, Sir Clement. Discussion of localized fibrous mesothelioma of pleura, 229
Prinzmetal, Myron (with Goldman, Alfred, Shaw, Clinton, Corday, Eliot, Fields, Joshua, Kennamer, S. Rexford, Smith, Allen, and Lindgren, Inga) Experimental methods for detection of changes of blood supply to heart, 105
Pulmonary emphysema, surgical management of (Crenshaw and Rowles), 398
  hypertrophic emphysema, localized (Williams), 522
  resections, use of Sk-Sd (Varidase) in management of early postoperative partial (Read), 284
  tuberculosis, resection in (Kelley and Pecora), 510
  tumors, induced (Burdette), 427
  venous occlusion, experimental (Hanlon, Sabiston, and Burke), 190

R

Rae, M. V. (with Taylor, H. E.) Endobronchial carcinosarcoma, 93
Ravitch, Mark M. Discussion of hemopericardium and constrictive pericarditis, 366
  Discussion of surgical treatment of carcinoma of throat, 255
  (with Bahnson, Henry T., and Johns, Thomas N. P.) Carcinoma of esophagus, 256, 269
Read, C. Thomas. Discussion of hemopericardium and constrictive pericarditis, 366
  Discussion of treatment of bronchopleural fistula, 582
  use of Sk-Sd (Varidase) in management of early postoperative partial pulmonary resections, 284, 303
Rehabilitation in thoracic surgery (Haas, Rusk, and Goodman), 304
Resection in pulmonary tuberculosis (Kelley and Pecora), 510
  of trachea for carcinoma (Cotton and Penido), 231
Robertson, Ross. Discussion of atelectasis and pulmonary blood flow in dog, 396
  Discussion of carcinoma of esophagus, 268
Rockey, Edward Ernest. Care of thoracic and thoracoabdominal wounds in combat zone in Korea, 435, 480
Roentgenogram in bronchography, indications for delayed (Kane and Heiser), 70

Roos, Albert (with Peters, Richard M.) Effects of atelectasis on pulmonary blood flow in dog, 389
Rowles, Donald F. (with Crenshaw, Gerald L.) Surgical management of pulmonary emphysema, 398
Rusk, Howard A. (with Haas, Albert, and Goodman, Walter N.) Rehabilitation in thoracic surgery, 304
Ryan, B. J. Discussion of nodular tuberculosis, 575

S

Sabiston, David C., Jr. (with Hanlon, C. Rollins, and Burke, Donald R.) Experimental pulmonary venous occlusion, 190
Sanger, Paul. Discussion of treatment of bronchopleural fistula, 583
Sarot, Irving A. Discussion of symposium on war wounds of chest, 479
Schmidt, Herbert W. (with Clagett, O. Theron, and McDonald, John R.), 213
Scott, George B. (with Litwak, Robert S., Gadboys, Howard L., and Ferrara, Joseph F.) Surgical approach for stenotic lesions of semilunar valves by excision and cusp replacement under direct vision, 165
Seley, Gabriel P. Discussion of rupture of esophagus, 387
Semilunar valves by excision and cusp replacement under direct vision, surgical approach for stenotic lesions of (Litwak, Gadboys, Scott, and Ferrara), 165
Shallenberger, Paul L. (with Fisher, Peter, Beck, William C., and DeWan, Charles H.) Concurrent lymphoma of lung and stomach—follow-up on previously reported case, 637
Shaw, Clinton (with Goldman, Alfred, Corday, Eliot, Fields, Joshua, Kennamer, S. Rexford, Smith, Allen, Lindgren, Inga, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105
Shefts, Lawrence M. Discussion of symposium on war wounds of chest, 474
Sherman, Roger T. (with Helmsworth, James A., Clark, Leland C., Jr., Kaplan, Samuel, and Largen, Thomas) Artificial oxygenation and circulation during complete by-pass of heart, 117
Shields, D. O. (with Chapman, John S., Jr., Carswell, James, Jr., and Wollenman, O. J.) Nodular tuberculosis, 568
Silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma (Kergin), 545
Sk-Sd (Varidase) in management of early postoperative partial pulmonary resections, use of (Read), 284
Smith, Allen (with Goldman, Alfred, Shaw, Clinton, Corday, Eliot, Fields, Joshua, Kennamer, S. Rexford, Lindgren, Inga, and Prinzmetal, Myron) Experimental methods for detection of changes of blood supply to heart, 105
Snyder, Howard E. Discussion of symposium on war wounds of chest, 474
Sommer, George N. J., Jr. Discussion of symposium on war wounds of chest, 477
Steele, John D. Rack for underwater drainage bottles, 323
Stenotic lesions of the semilunar valves by excision and cusp replacement under direct vision, surgical approach for (Litwak, Gadboys, Scott, and Ferrara), 165
Stephens, H. Brodie. Discussion of bronchiogenic carcinoma, a product of multiple sites of origin, 283
Discussion of surgical treatment of carcinoma of throat, 255
(with Grimes, Orville, F.) Surgical treatment of carcinoma of hypopharynx, larynx, and upper esophagus, 246
(with Thorburn, Jack D., and Grimes, Orville F.) Benign thymoma in hilus of lung, 540
Stomach, concurrent lymphoma of lung and (Shallenberger, Fisher, Beck, and DeWan), 637
Storey, Clifford F. Fibrosarcoma of bronchus—report of three cases diagnosed by bronchoscopy and treated by resection, 16
(with Umiker, William) Adenocarcinoma developing in a peripheral bronchial adenoma, 420
Strieder, John W. (with Aves, Fred H., and Desforges, Gerard) Mercury-bag bolus to expedite passage of esophageal dilator guide string, 34
Swift, Edward A. (with Grindlay, John H., and Clagett, O. Theron) Repair of tracheal defects with fascia and tantalum mesh; an experimental study, 482
Swindell, H. V. (with Murphy, James D., and Becker, Barney B.) Complications and results of treatment of bronchopleural fistula following resection for tuberculosis, 578

T

Taber, Rodman E. (with Ehrenhaft, Johann L.) Hemopericardium and constrictive pericarditis, 355
Tantalum mesh, repair of tracheal defects with fascia and (Swift, Grindlay, and Clagett), 482
Taylor, H. E. (with Rae, M. V.) Endobronchial carcinosarcoma, 93

Test, Pasquale S. (with Berman, Jacob K., and McArt, Bruce A.) Congenital esophagobronchial fistula in an adult, 493
Thomas, Colin G., Jr. (with Ziffren, Sidney E.) Healing of extensive cardiac wounds, 346
Thomas, Sir Clement Price. Discussion of localized fibrous mesothelioma of pleura, 229
Thoracic and thoracoabdominal wounds in combat zone in Korea, care of (Rockey), 435
aorta, traumatic saccular aneurysm of (Hollingsworth, Johnston, and McCooey), 325
approach to esophageal lesions, further discussion of right (Hollingsworth), 201
surgery, rehabilitation in (Haas, Rusk, and Goodman), 304
Thoracoabdominal wounds in combat zone in Korea, care of thoracic and (Rockey), 435
Thorburn, Jack D. (with Stephens, H. Brodie, and Grimes, Orville F.) Benign thymoma in hilus of lung, 540
Thymic neoplasms (Fisher, Coburn, and Effler), 58
Thymoma in hilus of lung, benign (Thorburn, Stephens, and Grimes), 540
Trachea for carcinoma, resection of (Cotton and Penido), 231
defects with fascia and tantalum mesh. repair of (Swift, Grindlay, and Clagett), 482
excision for carcinoma, trans-sternal (Minor), 88
Tracheobronchial trees of young rats, effects of increased endobronchial pressure, vitamin A deficient diets, and nasopharyngeal installations of bacteria on the (O'Neill, Bradshaw, Hightower, and Ward), 80
Tracheotomy, anterior mediastinal (Kleitsch), 38
Traumatic saccular aneurysm of the thoracic aorta (Hollingsworth, Johnston, and McCooey), 325
Tuberculous fibrinous bronchitis (Williams), 530
Tuberculosis, complications and results of treatment of bronchopleural fistula following resection for (Murphy, Becker, and Swindell), 578
nodular (Shields, Chapman, Carswell, and Wollenman), 568
resection in pulmonary (Kelley and Pecora), 510
Tumors, induced pulmonary (Burdette), 427

U

Umiker, William, and Storey, Clifford F. Adenocarcinoma developing in a peripheral bronchial adenoma, 420

V

Valle, A. R. Management of war wounds of chest, 457, 479
Vena cava with survival, wound of (Hudson), 101
Vineberg, Arthur M. Discussion of detecting changes of blood supply to heart, 115
Vitamin A deficient diets and nasopharyngeal installations of bacteria on the tracheobronchial trees of young rats, effects of increased endobronchial pressure (O'Neill, Bradshaw, Hightower, and Ward), 80

W

Wangensteen, Owen H. Discussion of carcinoma of esophagus, 267
Discussion of rupture of esophagus, 386
War wounds of chest, management of (Valle), 457
Ward, D. E., Jr. (with O'Neill, James F., Bradshaw, H. H., and Hightower, Felda) Effects of increased endobronchial pressure, vitamin A deficient diets, and nasopharyngeal installations of bacteria on tracheobronchial trees of young rats, 80
Waterman, David H. Discussion of rupture of esophagus, 386
Discussion of silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma, 566
Discussion of treatment of bronchopleural fistula, 585
Williams, Mark H. Acute tuberculous fibrinous bronchitis, 530
Discussion of symposium on war wounds of chest, 478
localized pulmonary hypertrophic emphysema, 522
Wollenman, O. J. (with Shields, D. O., Chapman, James S., Jr., and Carswell, James, Jr.) Nodular tuberculosis, 568
Woodruff, Warriner. Discussion of silicotic and tuberculosilicotic lesions simulating bronchiogenic carcinoma, 565
Discussion of symposium on war wounds of chest, 478
Wound of superior vena cava with survival (Hudson), 101

Y

Young, William P. (with Gale, Joseph W., Curreri, Anthony R., and Dickie, Helen A.) Plastic sponge prosthesis following resection in pulmonary tuberculosis, 587

Z

Ziffren, Sidney E. (with Thomas, Colin G., Jr.) Healing of extensive cardiac wounds, 346